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ROYAL COMMISSION OF INQUIRY INTO CERTAIN
DEATHS AT THE HOSPITAL FOR SICK CHILDREN AND
RELATED MATTERS.

Hearing held
8th floor
180 Dundas Street West
Toronto, Ontario

Roz: X
(Scot)

The Honourable Mr. Justice S.G.M. Grange	Commissioner
P.S.A. Lamek, Q.C.	Counsel
E.A. Cronk	Associate Counsel
Thomas Millar	Administrator

Transcript of evidence
for
August 16, 1983

VOLUME 19

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TORONTO, ONTARIO

1 ROYAL COMMISSION OF INQUIRY INTO CERTAIN
2 DEATHS AT THE HOSPITAL FOR SICK CHILDREN
AND RELATED MATTERS.

3

4

5 Hearing held on the 8th Floor,
6 180 Dundas Street West, Toronto,
Ontario, on Tuesday, the 16th
day of August, 1983.

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9

10 THE HONOURABLE MR. JUSTICE S.G.M. GRANGE - Commissioner
11 THOMAS MILLAR - Administrator
12 MURRAY R. ELLIOT - Registrar

13

14 APPEARANCES:

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M. HAYES)
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L. CECCHETTO) General of Ontario (Crown
Attorneys and Coroner's Office)
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R. BATTY) for Sick Children
M. THOMSON)
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21 B. PERCIVAL, Q.C.) Counsel for The Metropolitan
D. YOUNG) Toronto Police
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23 W.N. ORTVED) Counsel for numerous Doctors
K. CHOWN) at The Hospital for Sick
Children
24
25 E. MCINTYRE) Counsel for the Registered
Nurses' Association of Ontario
and 35 Registered Nurses at
The Hospital for Sick Children

(Cont'd)



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(b)

1 APPEARANCES: (Continued)

- | | | |
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| 4 | G.R. STRATHY)
E. FORSTER)
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Nurse |
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R.N.A. |
| 6 | J.A. OLAH | Counsel for Janet Brownless -
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| 7 | M. MANNING, Q.C. | Counsel for Mr. & Mrs. Gosselin,
Mr. & Mrs. Gionas, Mr. & Mrs.
Inwood, Mr. & Mrs. Turner, Mr.
& Mrs. Lutes and Mr. & Mrs.
Murphy (parents of deceased
children) |
| 8 | G.R. SOLOMON | Counsel for Mr. & Mrs. Hines,
parents of deceased child
Jordan Hines) |
| 9 | F.J. SHANAHAN | Counsel for Mr. & Mrs. Dominic
Lombardo (parents of deceased
child Stephanie Lombardo); and
Heather Dawson (mother of
deceased child Amber Dawson) |
| 10 | J. SHINEHOFT | Acting for Lorie Pacsai and
Kevin Garnet (parents of
deceased child Kevin Pacsai) |

VOLUME 19



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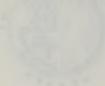
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---Upon commencing at 10:00 a.m.

3

4

5

6

MR. SOPINKA: Mr. Commissioner, I don't know whether this seating plan is designed to promote settlement, but I object to sitting next to the Attorney-General.

7

8

MR. PERCIVAL: He doesn't have to because my friends have arrived so he has to go some place else.

9

10

11

MR. SCOTT: The problem is,

12

13

Mr. Sopinka, everybody objects to sitting next to the Attorney-General but it had to be somebody.

14

15

16

17

THE COMMISSIONER: Well, I think we should all be greatly honoured by his attendance today.

18

19

20

21

Obviously we are just trying to work our way out, and all complaints will be heard in the course of the day or at the end of the day or any other time as to seating. I see now that Mr. Hunt is here with no seat provided for him.

22

23

24

25

If you think this is unfortunate all you have to do is contemplate what would have happened if we had been up on the 21st floor where they wanted to put us.

26

27

28

29

Well now, Mr. Lamek, you have a few more questions, I believe, have you not?

30

31

MR. LAMEK: Mr. Commissioner, before we begin again with Dr. Rowe I think we should perhaps



1

2

first for the record thank the Chairman of the
Municipal Board for permitting us the use of the
room and at the same time, sir, acknowledge your
advocacy skills in persuading him to do that.

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first for the record thank the Chairman of the
Municipal Board for permitting us the use of the
room and at the same time, sir, acknowledge your
advocacy skills in persuading him to do that.

Second, in the course of the two

weeks since we were last sitting, Mr. Shinehoft
has kindly provided to us his copy of the Pacsai
material, and replacement pages of some portions of
that chart have been prepared and distributed; in
particular, pages 33, 42, 45, 46 and 77.

As I say, replacement pages have been
distributed to counsel, sir, and perhaps at the end
of the day we can retrieve the exhibit copy and
replace them in that as well.

THE COMMISSIONER: What is the number
of that exhibit?

MR. LAMEK: The Pacsai exhibit?

I am sorry.

THE REGISTRAR: The Hospital record?

MR. LAMEK: Yes.

THE REGISTRAR: 106.

MR. LAMEK: 106. Thank you.

Next, Mr. Commissioner, the Hospital
has furnished to us copies of final autopsy reports
that do not appear in the bound Hospital charts that



1

2 have already been marked as exhibits.

3 We have had those prepared in a
4 separate binder with numbered pages, and I would ask
5 that that binder of final autopsy reports be the next
6 exhibit, and copies of that binder have been provided
7 to counsel.

8

THE COMMISSIONER: Exhibit 124, and
will you tell me which babies?

9

MR. LAMEK: Yes, sir. They are
10 Woodcock, Perreault, Taylor, Dawson, Hoos, Turner,
11 Shrum, Monteith, Velasquez, Gage, McKeil, Volk,
12 Lutes, Onofre, Gosselin, Belanger and Floryn.

13

THE COMMISSIONER: Thank you. That
is the final autopsy report for all the children ---

14

MR. LAMEK: Who were autopsied, yes.

15

THE COMMISSIONER: Yes. All right.

16

17 ---EXHIBIT NO. 124: Final Autopsy Report re Babies
Woodcock, Perreault, Taylor,
Dawson, Hoos, Turner, Shrum,
Monteith, Velasquez, Gage,
McKeil, Volk, Lutes, Onofre,
Gosselin, Belanger and Floryn.

18

19 MR. LAMEK: Mr. Commissioner, at
the end of our last hearing I had thought I had
completed my examination of Dr. Rowe, and I have
spoken to Mr. Scott, and with your permission, sir,
I have just a very few more questions of Dr. Rowe

20

21

22

23

24

25



1

2

with respect to a matter of which I learned only
3 last week.

4

THE COMMISSIONER: Yes.

5

6

MR. LAMEK: It will not take very
long, and perhaps we could ask Dr. Rowe to come back.

7

THE COMMISSIONER: Yes. All right.

8

MR. SCOTT: You couldn't save these
for reply?

9

10

MR. LAMEK: No. I think everybody
should know about it.

11

12

MR. ORTVED: If I may just have your
indulgence, Mr. Commissioner?

13

THE COMMISSIONER: Yes.

14

MR. ORTVED: Thank you, Mr. Commissioner.

15

DR. RICHARD DESMOND ROWE, Resumed

16

DIRECT EXAMINATION BY MR. LAMEK: (Continued)

17

Q. Dr. Rowe, first to complete

18

the matters that we were talking about when last we

19

met, you had given to me at the close of your

20

evidence a list of six names in addition to that of

21

Justin Cook, and those were the children who, as I

22

understood you, and subject to the resolution of any

23

pharmacological debate and dispute that may exist,

24

were those who in your judgment were most likely to

25

have died as a result of a digoxin overdose.



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I understand that there is an additional name that you omitted to give me in that list.

A. Yes. There is, Mr. Lamek.

That is the name of Lombardo.

Q. Yes.

A. And I appreciate the opportunity to bring that in.

Q. Not at all. Thank you very much, Doctor.

Now, Doctor, one thing has come to my attention since last we met and I think we can deal with it very rapidly. We discussed in the course of your evidence in chief Mortality and Morbidity Conferences that were held in September, 1980, and then the meeting that was held ~~in~~ January, 1981, ~~and~~ following a review of some twenty deaths.

A. Yes.

Q. And at that stage as I understood all ward deaths prior to December 31, 1980 had been reviewed by your group?

A. Yes.

Q. Now I understand, Doctor, that you had scheduled a further review to be done with respect to deaths from and after January 1st, 1981, and that that review was scheduled to begin on Monday,



1

2

March 21st, 1981.

3

A. No, I don't believe.

4

Q. You don't have a recollection?

5

A. No.

6

Q. You have told us that the police investigation which, of course, was set in train on the weekend of March 21 and 22 ---

7

A.. Yes.

8

Q. --- overtook any investigations by the Hospital?

9

A. Yes.

10

Q. But you have no recollection of having arranged for a further review of the 1981 deaths to take place at the end of March?

11

A. No, not prior to the end of the time of that weekend. I think at the end of the weekend obviously there was going to have to be examination of issues in connection with those deaths, but that was taken by the police study.

12

Q. Do you have any recollection on March 21, which I believe was the day when you met with the coroners?

13

A. Saturday.

14

Q. Saturday the 21st would be the day.

15



1

2

3

A. Saturday the 21st would be

the day.

4

5

6

7

Q. Do you have any recollection
at that time of saying to Dr. Teperman that in fact
a review had been arranged of the post January 1
deaths, and was to begin on the following Monday?

8

A. I can't recall.

9

Q. Then there is not much point
in my asking you any questions about that, Doctor.

10

A. No. I'm sorry.

11

MR. LAMEK: Thank you, Doctor.

12

THE COMMISSIONER: Yes. Thank you.

13

Mr. Scott?

14

MR. SCOTT: I think I will come
from here, Mr. Commissioner, it will be easier,
because I am going to need some help.

15

THE COMMISSIONER: Yes. All right.

16

Whatever is convenient.

17

MR. SCOTT: First of all,

18

Mr. Commissioner, I have placed on the ---

19

THE COMMISSIONER: I am not sure -

20

I can hear you. Can everyone hear you?

21

MR. SCOTT: This is not usually a
problem for me.

22

THE COMMISSIONER: No. But I am

23

24

25



1

2

just wondering whether that microphone is actually
working.

4

5

MR. SOPINKA: Well, I can't hear,
but I am not listening.

6

MR. SCOTT: Not yet.

7

THE COMMISSIONER: You don't know.

8

Well, I think it is probably working.

9

Let's try and if anyone can't hear just raise his
hand and we will do something about it.

10

11

The acoustics ~~is~~ ^{are} supposed to be very
good in here. I don't know whether they are or not.

12

13

MR. SCOTT: They are excellent,
almost everybody is inaudible, including the witness
and the Commissioner.

14

15

I should say, Mr. Commissioner, that
I put on the board what is really a series of graphs
which I undertake to prove when my opportunity, some
time late next year, comes to call evidence, but
because I may want to ask some questions about it I
tender it now as an exhibit, and perhaps it should
be given a number to be proved. And I want to explain
to you what it is and as I say, I undertake to prove
in due course the manner in which it has been
accomplished.

23

24

The data for the graph is based on the

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monthly death reports which are compiled by the census control clerk at the Hospital. Those monthly reports are based upon a daily census sheet which is completed for each ward or place in the Hospital by the head nurse in charge of that ward or place, and if a patient has died the census control clerk checks for the last ward where the patient was alive in order to get the geographical place of death.



1
15aug83 2
BMcra

Now, in the left-hand margin, you
will see --

4 THE COMMISSIONER: Before we get
any further, you want to give it a number, I under-
5 stand?

6 MR. SCOTT: Yes.

7 THE COMMISSIONER: Exhibit 125.

8 ---- EXHIBIT NO. 125: Mortality Chart.

9
10

11 THE COMMISSIONER: All right, the
left-hand side.

12 MR. SCOTT: Q. The left-hand side
13 are the number of deaths rated in multiples of five
14 and numbered in multiples of ten from zero to fifty.
15 Along the bottom, indicated by each point on the
16 measured line, are months from January 1, 1976 to
17 December 31, 1982. 'J' indicates January, the start-
18 ing point of each year. The black line at the top
19 of the page is the total deaths per month for that
20 period that occurred anywhere in the hospital.

21 So, at the top line, with the peaks
22 and valleys that it reveals, is a monthly accounting
23 of how the total number of deaths in the hospital
24 varied, and you will see, in examining that, that there

25



B2

1

2 are dots which represent the months and there are
3 peaks and valleys and so on illustrated on that chart.

4

5 I am told that there is an orange
line, and I don't see the orange line - it's a brown
6 line. What is the brown line, neonates?

7

8 Yes, the brown line is, as you see,
Ward 7G, which is the neonate ward.

9

10 THE COMMISSIONER: I'm sorry, which
is the brown line?

11

MR. SCOTT: Here (indicating).

12

13 THE COMMISSIONER: No. I know what
it is but what does it represent?

14

15 MR. SCOTT: You know what it looks
like, you know what brown looks like.

16

17 THE COMMISSIONER: Yes. But what
does it represent?

18

19 MR. SCOTT: It represents the neonate
ward, which is --

20

21 THE COMMISSIONER: You have told us
the brown line, but it seems to be over a -- oh, there
is a yellow line as well?

22

23 MR. SCOTT: Well, we're coming to
that.

24

25 THE COMMISSIONER: Yes. All right.

26

27 MR. SCOTT: The brown line is the

28



1
B3 2 neonate ward, No. 7G, and you will recall - I think
3 it is in evidence - that that is a neonatal intensive
4 care unit in which the age limit essentially is less
5 than one month of age.

6 Again, the number of deaths in that
7 ward is plotted on a monthly basis, and you will see
8 the degradations from month to month over this
9 period.

10 The next line is the all-cardiac
11 line.

12 THE COMMISSIONER: And that colour
13 is?

14 MR. SCOTT: And that colour is red -
15 it used to be orange - and that records, on a monthly
16 basis, all cardiac deaths - I think I've got the
17 cardiac line - yes, all cardiac deaths which occurred
18 in the hospital, and that will include cardiac deaths
19 in 4A and 4B. Again, it is plotted out on a monthly
basis.

20 The purple line --

21 THE COMMISSIONER: I take it the
22 only places, though, they could be would be, presumably,
23 in the operating room or in the intensive care unit?

24 MR. SCOTT: No. I will come to that.

25 MR. MANNING: I'm sorry, Mr.



B4

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Commissioner, I can't hear you back here.

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THE COMMISSIONER: Well, they promised me that this - no matter what I did to it - would record what's going on, but it obviously doesn't. We will have to work on that.

Can you hear me now?

MR. MANNING: Yes.

THE COMMISSIONER: I guess I just start to mutter. My trouble is, when I'm not too sure what I'm saying, I mutter and, when I have some idea what it's all about, I yell!

All right now.

MR. SCOTT: I just want to make a note of that!

THE COMMISSIONER: The cardiac deaths, I take it, will be all over the hospital?

MR. SCOTT: Yes.

THE COMMISSIONER: Not just in the operating room?

MR. SCOTT: They will be dominantly, I think, in the cardiology section, but they are all over the hospital.

The purple line is all other deaths, excluding cardiac deaths.

MR. SOPINKA: Where is the purple



1
B5 2 line?

3 MR. SCOTT: Here.

4 THE COMMISSIONER: Well, the sum
5 then of cardiac and other I take it would equal
6 the red plus purple would equal brown; is that right?

7 MR. SCOTT: Plus ICU.

8 THE COMMISSIONER: The yellow line,
though, we haven't accounted for that yet.

9 MR. SCOTT: What you have is all
10 deaths are geographic; that is to say, because they
11 occurred in the Hospital, they are plotted on that
12 line. The brown line, 7G, is geographic, too. You
13 are only plotted on that line if your death occurred
14 in the neonatal ward of whatever cause.

15 THE COMMISSIONER: That's right.

16 MR. SCOTT: All cardiac is not
17 geographic. That's a diagnostic line, and I will
18 explain how that was computed in a second. Let me go
to ICU.

19 ICU, the yellow line, is again a
20 geographic line in the sense that the death has to
21 have occurred in the ICU before the death is plotted
22 on this line.

23 THE COMMISSIONER: The purple line,
which is All Others means all others of what?

24
25



1

2 MR. SCOTT: Let me come to that in
3 a moment.

4 The blue line at the bottom is 5A
5 and then, in 1980, turning into 4A and 4B, and that
6 again is a geographic line, you had to be on that
ward to figure in that line.

7

THE COMMISSIONER: All right.

8

9 Now, help me out with the purple
line then. What's that?

10

11 MR. SCOTT: First of all, if you add
12 up all the other lines which are geographic lines;
13 that is, 5A, ICU, All Other and 7G, you will get
14 the All Deaths line, the black line. In other words,
if you add up all the geographic lines, you will get
this total.

15

16 THE COMMISSIONER: All right. There
is a 5A line, and that's the green line, is it?

17

MR. SCOTT: Yes.

18

19 THE COMMISSIONER: Isn't that, the
one at the bottom, isn't that green?

20

MR. SCOTT: Aquamarine.

21

THE COMMISSIONER: I'll settle for
green.

22

MR. LAMEK: Turquoise.

23

MR. SCOTT: Trust Mr. Lamek!

24

25



1

2 THE COMMISSIONER: I just want to
3 make sure I can recognize the blue line when I see it.
4 Could you point out the blue line
5 for me, please.

6

MR. SCOTT: Here

7

THE COMMISSIONER: Oh, I thought
that was the green line. That is the blue line?

8

MR. SCOTT: Yes.

9

THE COMMISSIONER: That is 4A, or
is it 5A? It says '5A'.

10

MR. SCOTT: Well, it begins, as you
will recall, as Ward 5A.

11

THE COMMISSIONER: Oh, I see.

12

MR. SCOTT: And then, I think in
14 April of 1980, the 1st of April, they moved from 5A to
15 4A and 4B, and that is recorded here, just so you will
16 have a note of it.

17

THE COMMISSIONER: So, whether we
call it green or blue or aquamarine or anything, that
is all of 4A and 4B and 5A?

18

MR. SCOTT: That's right, yes. And
20 the All Other line is all other deaths, excluding
21 cardiac deaths and excluding the others noted. That
22 is, if you exclude cardiac deaths, ICU deaths, 4A-4B
23 deaths, you then produce the All Other line.

24

25

It certainly was so on 4A/D
as between Oct 1/79 - June 30/80
and July 1/80 - March 31/81!

- ① Check that this is so
- ② In any event, the ICU is one of the places where patients do, traditionally die.
- ③ MAY be corroborative of the "severe clustering" hypothesis EXCEPT that the Ward 4A/D → ICU transfers were v. few and therefore the jump in ICU death rate ^{may} ~~must~~ reflect a higher incidence of very ill patients in OTHER areas of the Hospital.
- ④ ? Time clustering of ICU deaths?



1

2 That is presented so you won't think,
3 Mr. Commissioner, that anywhere in this Hospital,
4 contrary to what has been reported, there is a 60 per
5 cent increase that attaches to the Hospital death
6 rate. That simply isn't so, as this chart -- a 600
7 per cent increase. That simply isn't so, as the chart
8 reveals. And, for example, we'll be dealing with
some particular points.

9 If you look at this chart, for
10 example, if you look at July and August of 1980, the
11 two opening months of what we call the epidemic
12 period where there is an increase of deaths in this
ward, you will see that there is an extravagant
13 increase of a much greater proportion in ICU deaths.
14 So that if one, in July or August of 1980, was looking
15 to find an area in the Hospital where there were an
16 escalated number of deaths, you would be attracted
17 not so much by 4A and 4B but, rather, by the ICU in
that case.

18
19 This is simply presented so that you
will have a complete picture and will not be obliged
20 to look only at one ward without relation to the
others.

21 // For example also, when you come to
22 March, the last month of the epidemic period, where
23 It is helpful to do this (but not helpful
24 to the Hospital's position) because the chart
25 demonstrates that all the dramatic month-to-
month fluctuations in mortality were quite common -
place in other areas, THEY WERE NOT ON 4A/B!

Why were children not being sent from
4 A/I to the ICU?

2 possible explanations are:

(a) the ICU, because of an overload,
was refusing to accept patients
(as to which the Hosp. will
have to adduce ev.)

(b) 4 A/I patients were not, until
immediately prior to their death,
considered to be in need of
intensive care — and when,
with the sudden onset of terminal
events, the need did become
clear, the decision was too rapid
to permit transfer to the ICU
except in the case of Paesai.



1

2 there is an increase in 4A-4B, there, you will see
3 that there is a decline in the ICU rate, and we'll
4 have something to say about that in due course.

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DM/wb

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2 Then in January, of course, in 1982, there is an
3 extravagant increase, the greatest ever I think in
4 history, in ICU deaths.

5 For example, I think the increase that
6 occurred in ICU deaths in 1981-1982 is far greater in
7 a gross or percentage sense than any increases that
8 occurred in this period in 4A/4B. So we wouldn't want
9 you or any members of the public to get the sense that
10 the death rate in the hospital is an evenly graded
11 exercise. It is represented by elaborate shifts which
12 are sometimes difficult to explain, and which we will
try and deal with, at all levels.

13 Now, as I said, I undertake in due
course to prove that.

14 THE COMMISSIONER: Could we also have
15 copies of it?

16 MR. SCOTT: Yes sir, we are having
17 copies made. I should also say that we will have
18 this afternoon, I thought it would be here this
19 morning, but it can't be, a bar graph over the same
20 period which I will seek to introduce which will show
21 some other factors about mortality rates in the
hospital.

22 EXAMINATION BY MR. SCOTT:

23 Q. Now, Dr. Rowe, about half-way
24 through your evidence, you were being asked, by Mr.

25 Lamek,



C2

1
2 whether the deaths of certain babies that occurred
3 on -- you were asked to describe, to give your
4 opinion as to the cause of the deaths of certain
5 babies on 4A/4B in the epidemic period. Having done
6 so, you were in almost every case asked, following
7 that, by Mr. Lamek, if the death was consistent with
8 digoxin toxicity. You gave your answer, and I think
9 if I can summarize correctly, the upshot of most of
10 your answers until we come down to Baby Pacsai, was
11 that the manner of dying was consistent with digoxin
12 toxicity in some of those cases, but there was no
13 evidence indicative of it until the Baby Pacsai.
14 Have I summarized that correctly?

15 A. Yes, I think so.

16 Q. And, of course, Baby Pacsai is,
17 I think, the first baby in which there was a premortem
18 serum digoxin level of significant elevation, would
19 that be fair?

20 A. Yes. Apart from, I think, one
21 sample in one of the earlier babies.

22 Q. Well, the premortem serum
23 sample for Pacsai was taken, as I have it, on March
24 10th, and was 10 nanograms?

25 A. Yes.

Q. And I take it that while there

Because the level was > 4.7 and because
one did not know how much
higher than 4.7, how could this
be merely "of a little concern"?

What of 3.5 ()
4.7 () ?



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had been previous ~~pre-s~~ ^{post} samples at the three and four level, this was the first premortem sample of this kind of elevation in the epidemic. Also I am just trying to check the sample of Estrella? Estrella was 4.7, premortem?

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8
A. I am not sure if it was
greater than 4.7.

9
Q. Yes.
10
A. So it was a level about this
that was certainly of a little concern.

11
12
Q. All right, let's leave it this
way then. That prior to Estrella, were there any
13 premortem samples which gave you any particular
concern?

14
15
A. No.

16
Q. And between Estrella and Pacsai
17 were there any premortem samples that gave you any
particular concern?

18
A. Can I just check that one
point?

20
21
Q. Yes. I can tell you, there was
Fazio 1.5; there was Floryn 2.1; Leith 2.1; Gionas 1.2;
22 Manojlovich -- I think I have the name right, 2.2?

23
A. Yes.
24
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This statement: He was asked, re
end death

- Was death, inc terminal events and then
answ - hopes consistent w.
patient's clinical condition
- Also consistent w. diag infrx^2



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Q. And all those samples would
be within, all those premortem samples would be
within the normal therapeutic range?

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A. Yes.

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Q. So I have it then that Estrella
is a cause for concern for reasons that you have given,
but between Estrella and Pacsai would I have it right
that there are no other cases that cause you concern
with respect to digoxin toxicity?

A. No, not after examination of
the levels and the time of the sampling.

THE COMMISSIONER: Excuse me, Mr.
Scott, what is the number of the preliminary inquiry
exhibits, it has a number I know?

MS. CRONK: It is Exhibit 32.

THE COMMISSIONER: Thank you. I am
sorry.

Q. Dr. Rowe, the way your evidence
in chief went, by virtue of the kind of questions you
were asked, seemed to suggest that there were only
two possibilities as the underlying cause of death,
that is cardiac arrest of some type, or digoxin
toxicity. Indeed, when I said there might be others,



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it was suggested that no one had suggested any others.
So what I want to deal with first, at some considerable
length, is what I have called as a heading "The
Mechanics of Babies Dying". Let me put a few
propositions to you, just to get started, to see if
you and I agree, and we should, because I think you
told me most of this.

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8 First of all, the stoppage of a heart
9 is the characteristic final event of all deaths, is
10 that correct?

11 A. Yes.

12 Q. And that is cardiac arrest,
13 the stopping of the heart?

14 A. Yes.

15 Q. So that what happens when the
16 heart stops is that the muscle of the heart stops
17 pumping, traditionally?

18 A. Yes.

19 Q. Or characteristically, is that
right?

20 A. That is right.

21 Q. And whether the muscle of the
22 heart has stopped pumping or not can be monitored and
23 observed by an electrocardiogram?

24 A. You can see where there is no

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Cute but
bush!



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further electrical activity of the electrocardiogram, and the pulse and the blood pressure measurements would tell you that it is not pumping.

Q. And I take it that the --

THE COMMISSIONER: Could I just have that again, because there seems to be a distinction that I haven't got.

THE WITNESS: The electrical activity, there may be electrical activity on the electrocardiogram of some sort when there is still -- when there is no action of the pump itself. So that it doesn't necessarily mean you have to have no activity of the electrocardiogram for the patient to be dead.

THE COMMISSIONER: But I take it, it means something.

THE WITNESS: It is very close.

THE COMMISSIONER: So the electrocardiogram will show a slowing of the heart, perhaps not a total stoppage.

THE WITNESS: Yes.

THE COMMISSIONER: Is that what you're saying?

THE WITNESS: Yes, but you still may have some electrical activity when the heart is not acting as a pump. So one has to use other measures



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such as the pulse and the heart beat and the blood pressure.

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Q. But it can be physically observed as well, can it not?

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A. Yes.

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Q. All right. Now, heart stoppage, or cardiac arrest, you have told us is the characteristic final event. I put it to you, if a man was stabbed in the back with a knife, the characteristic final event which caused his death would probably be cardiac arrest, the stoppage of his heart?

A. Yes.

Q. And that would be so if he suffered from blood poisoning in the right toe and was going to die, that cardiac arrest, or heart stoppage, would again be the characteristic final event that signified his death?

A. That would be so.

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Q. The stopping of the heart?

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A. Yes.

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Q. Yes. And there is ---

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THE COMMISSIONER: I am sorry. I

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have been through some of this before. I thought
that at some point the brain damage becomes so bad
that even though the heart keeps going the medical
fraternity consider that clinical death.

7

THE WITNESS: Yes. That can be ---

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THE COMMISSIONER: Am I wrong?

9

THE WITNESS: No, that can be true
too.

10

MR. SCOTT: But the heart is still
pumping or may be still pumping.

11

THE COMMISSIONER: If the heart is
still pumping, and I don't know, we may just be
fussing about semantics.

12

THE WITNESS: No, I don't think it
is fussing, but that is a special category. I think
that is different from most situations.

13

MR. SCOTT: The reason I am concerned
about this, Mr. Commissioner, is we have thought about
cardiac arrest as the cause. That is to say the
underlying cause of death. In fact it may be the
underlying cause but there may be, and I have a list

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of 14 other underlying causes which lead to cardiac
arrest properly understood; that is the final stoppage
of the pump action.

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THE COMMISSIONER: Yes. If I understand the various theories on this, the causes of death that have been advanced so far are heart disease and digoxin poisoning.

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MR. SCOTT: There are 13 more.

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THE COMMISSIONER: The symptoms of

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both, I suppose, could be cardiac arrest.

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MR. SCOTT: Well, we will be coming to that.

THE COMMISSIONER: Yes. Well, at least not the symptoms so much as the demonstration.

But the cause of death I don't think anybody has said has been cardiac arrest.

MR. SCOTT: Well, I ---

THE COMMISSIONER: Or at least I

haven't heard that.

MR. SCOTT: Well, perhaps if I can

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ask you just this once, Mr. Commissioner, to be patient.

THE COMMISSIONER: Yes.

MR. SCOTT: Around about November you can lay into me seriously and tell me to stop,



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but I would really like to deal with ---

THE COMMISSIONER: The only reason I am doing that is not to demonstrate anything but my own ignorance. When I don't understand, that is when I ask the question.

MR. SCOTT: I am very grateful for those interventions.

Q. Now, Dr. Rowe, do I understand from the evidence you have previously given that there are essentially two types of heart stoppage? The first is when the heart simply stops pumping; that is when there is no contraction at all in the muscle.

A. Yes.

Q. Yes. And the second is where the contraction continues but is ineffective to pump sufficient blood out of the heart.

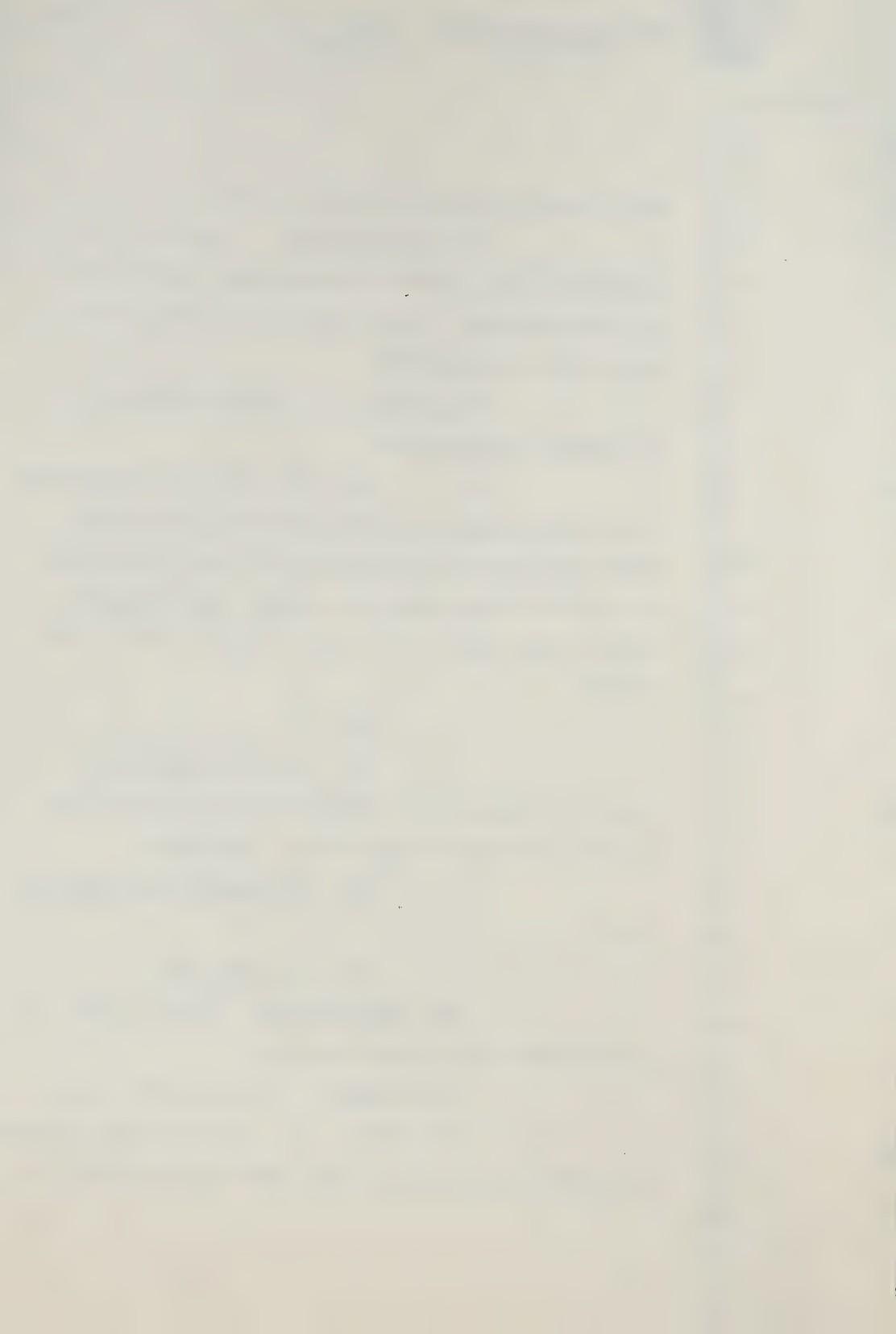
A. Yes. In ventricular fibrillation.

Q. So that is ---

THE COMMISSIONER: That is what is called ventricular fibrillation?

THE WITNESS: Fibrillation, yes.

MR. SCOTT: Q. So the heart stoppage or cardiac arrest as a final event causing death is





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typical of two types: one in which the muscle contraction ceases altogether, and the second in which the muscle contraction continues but is not sufficiently powerful to pump blood out of the heart in sufficient quantities?

A. Yes. And eventually that type becomes an arrest too.

Q. Yes. And what happens in the fibrillation case is that there is a loss of oxygenation in the other parts of the body? They have no nourishment and those body parts die.

A. Yes.

Q. Now one other term that we have dealt with and perhaps it is obvious, but resuscitation, and tell me if I am correct about this, is an effort by physical means, drugs or perhaps otherwise to get effective contractions underway so that they are self-sustaining?

A. Yes, that is true.

Q. So when you have a heart stoppage or a cardiac arrest which would be the final event, unattended, of all deaths, resuscitation is introduced in appropriate cases to mechanically or chemically or by some other means get the contractions to begin again?



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A. Yes, that is the intent, yes.

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Q. Now when that fails and the heart can't be induced to pump again, the patient is dead?

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A. Yes.

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Q. And when the patient dies a question asked is what caused the patient to die? That is the layman's question, isn't it?

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A. Yes, it is.

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Q. And is that for a doctor the same question as why did the heart contractions or effective heart contractions stop?

13

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A. Yes. That would be the question we would ask.

15

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Q. So when you have a cardiac arrest you don't assign cardiac arrest as the cause of death; you (though it is technically the last moment) you ask what caused that cardiac arrest?

18

19

A. Yes.

Q. Am I right?

20

A. You are right.

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Q. And I take it that that observation that the critical question, "What caused the cardiac arrest by which we will all die? What caused it?" is as appropriate for babies as it is



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for adults?

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A. Yes, it is.

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Q. Now having said that, I want
to take you to some of the ways cardiac arrest can
be caused, and I want you to think in this instance
particularly of babies and particularly in this
Hospital and the so called epidemic period.

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I want to see if I can't get, with
your help, a list of the possible causes that led
heart contractions to stop or caused heart cardiac
arrest typically in this Hospital, and indeed at the
end I may ask you to give us (I don't think you have
prepared it yet, but you are going to be around for
a while) a list of the 36 babies assigning a cause
to the cardiac arrest which led in most cases to
their death. In all cases to their death.

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Well let's deal first of all with

heart failure. Is heart failure a cause or a
potential cause of cardiac arrest?

A. Yes, it is.

Q. And what does a doctor mean
when he talks about heart failure?

A. Well, he means that the state
of the heart contractions have reached a point where
they cannot adequately pump blood around the body.



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Q. Yes. And is that characteristically a phenomenon in which the muscle of the heart tires out and stops?

5

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A. That is the simplest way of putting it.

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Q. Yes. And that tiring out of the muscle of the heart in heart failure, is that characteristically a result of stress or pressure created by abnormal construction of the heart?

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A. Usually - it may occur from infection of the heart muscle, but it is something that affects the performance of the muscle cells of the heart, yes.

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Q. All right. Now two weeks ago when we had the charts of the various babies on the lectern, you led us through some babies who had hearts that were abnormal in physical structure.

17

A. Yes.

18

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Q. And indeed I think most of the babies in your Ward 4A and 4B in the epidemic period did, did they not?

21

22

A. I think there were only three who did not have abnormal hearts.

23

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Q. All right. Now apart from anything else is a baby with a heart structure which

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is abnormal or defective a candidate for heart
failure?

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A. Particularly - that is true
particularly if the malformation is not a very mild
one.

7

Q. All right.

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A. Unless it is mild they are
candidates for failure.

9

Q. So that that baby with an
abnormally structured heart will die like all babies
do because of cardiac arrest, but one of the potential
causes for the cardiac arrest is heart failure,
characteristically, the abnormal structure of the
heart and the fact that the muscle tires under the
stress of that abnormality?

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A. Yes. The mode may be a
little different for different malformations, but
heart failure in the end is a good general term for
what takes place in that situation.

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THE COMMISSIONER: Could I interrupt
for just a moment? What are the external symptoms of
the difference between the ventricular fibrillation
and the true stoppage of the heart?

THE WITNESS: There is no external
difference. The only way you can tell that is by the



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electrocardiogram. Unless the chest were open and you were actually looking at the heart in the gross form.

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THE COMMISSIONER: When in these medical records they refer to ventricular fibrillation as being observed, they can't be sure?

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THE WITNESS: They can be sure if they see the electrocardiogram but not otherwise. The electrocardiogram is specific for ventricular fibrillation.

11

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THE COMMISSIONER: I see. All right. So it will show some ---

13

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THE WITNESS: It will show a very definite and consistent characteristic rhythm abnormality.

16

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18

MR. SCOTT: Q. So that a baby that has a grossly deformed septum, for example, an abnormal structure of the heart, may be a candidate for heart failure strictly speaking?

19

A. Yes.

20

21

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Q. That is the mechanical stress on that defective organism may lead simply to its wearing out?

23

A. Yes.

24

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Q. Now at this point I want to



D10

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2 stop because in every one of these deaths - I shouldn't
3 say every one but in close to every one Mr. Lamak,
4 having got you to give your opinion as to the death,
5 sought to invite you to compare the death of Baby X
6 or Baby Y in terms of the presence of bradycardia,
7 vomiting, the sudden deterioration or onset,
8 ventricular fibrillation, arrhythmia and shallow
9 respiration. And you may recall his point was, well,
10 Baby X appeared to be stable and then these things
11 happened, and that distinguishes that death from
the others.

12 Do you remember the line of questioning
13 that he put to you in each case?

14 A. Yes.

15 Q. Yes. All right. Now I want
16 to ask you about those.

17 Dealing with the first cause of heart
18 stoppage, heart failure, and before I do I would
19 like to read you evidence given by Dr. Ralph Kauffman
20 at an inquest into the death of a baby, Garry Murphy,
21 who died outside the epidemic period and after the
epidemic period and whose serum revealed digoxin
22 toxicity. Readings beyond the therapeutic range.

23 Now, first of all, do you know who
24 Dr. Kauffman is?

25



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A. Yes, I do.

3

Q. And I take it he is a well
known pharmacologist?

5

A. Yes, he is.

6

Q. In Chicago?

7

A. Yes. Detroit.

8

Q. Detroit?

9

A. Yes.

Q. And he was asked and he was
called I can tell you by the Crown in this case
and he was asked ---

THE COMMISSIONER: Was this the
inquest?

MR. SCOTT: This is the inquest.

Q. He was asked about the signs
of digoxin toxicity.

MR. PERCIVAL: What page is that?

MR. SCOTT: It is page number 12 of
the Kauffman excerpt. I don't think it is page 12
of the entire transcript.

Q. I just want you to listen to
the question and answer and then I'm going to ask
you if you agree with it or have any comment to make
about it.

"Q. The signs of toxicity,



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"especially in an infant, if you had
vomiting which we have heard from a
previous witness, Dr. Hesslein, poor
feeding, irritability, are these
necessarily things which would hold
up a red flag to you and say ah-ha,
there is digoxin toxicity present?"

The doctor says:

"I have to respond to that by saying
that the symptomatic signs of digoxin
toxicity in infants are rather non-
specific, and usually are symptoms
that can be due to other factors, and
it is difficult in many situations in
a clinical situation to be certain
whether or not a specific symptom is
due to or not due to digoxin in a
child. And this is where levels come
in handy sometimes to help you sort
that out. Vomiting, it is true that
vomiting, loss of appetite, irritability,
can be symptoms associated with toxic
digoxin effects. They can also be
associated with a myriad of other
things in infants this age, and that
is why it is so difficult to make a
definite association."



16aug83 2 Do you agree with that statement?

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BMcra 3 A. Yes, I do. I don't think
4 there would be any difficulty in any pediatric cardio-
5 logist agreeing with that statement.

6 Q. All right. So that, when
7 Mr. Lamek asks you, in the case of the death of Baby X,
8 well, wasn't there vomiting here, is it possible that
9 vomiting, as Dr. Kauffman says, can be attributable to
digoxin toxicity as well as a myriad of other causes?

10 A. Yes.

11 Q. All right.

12 A. We have examples of babies
13 who vomited in this group where we know that the
14 digoxin levels were absolutely within the normal range.

15 Q. All right.

16 Let's take then -- we are dealing
17 with the first cause of heart stoppage, which is
18 heart failure, a functional abnormality in the heart,
19 and I want to go through the things that Mr. Lamek
asked you about.

20 When you have a case of heart
21 failure, you will typically have an abnormality of
22 the heart structure?

23 A. An abnormality or an infection.

24 Q. Of the heart structure?

25



Rowe
ex. (Scott)

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E2

A. Of the heart structure, yes.

Q. Yes. Is it accompanied by
bradycardia from time to time?

A. It may be.

Q. Yes. Is it accompanied, as
death approaches, with vomiting from time to time?

A. Yes, especially if the failure
is bad.

Q. Yes. Is the terminal event
often a sudden onset?

A. Yes.

Q. Is it accompanied by ventricular
fibrillation from time to time?

A. Yes. There is a proportion
that do that.

Q. Yes. Is it accompanied by
arrhythmia?

THE COMMISSIONER: I'm sorry, let us
pause there for a moment.

Is ventricular fibrillation, is that
a progress towards the stoppage of the heart?

THE WITNESS: Yes. It is a mode of
the heart stoppage, but it is not necessarily present
in every baby who dies.

THE COMMISSIONER: Well, I am still

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Rowe
ex. (Scott)

E3

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2 having a little trouble in distinguishing between them.

3 Obviously, the heart stoppage, I can
4 understand with no difficulty at all, but ventricular
5 fibrillation, I am having some problems. I know the
6 result of it is you don't get enough blood around the
7 rest of the body.

8

THE WITNESS: Yes.

9

THE COMMISSIONER: But what does it
mean with the heart? What's happening?

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THE WITNESS: Well, it's an end stage
situation; the heart is just --

12

THE COMMISSIONER: Barely beating.

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THE WITNESS: Has a chaotic obstruction. I think it has been referred to in previous statements by pharmacologists in some of these hearings I am not sure whether it is the inquest or not - that the heart looks like you had a bag of worms in your hand or you had a handful of worms and you see very chaotic but ineffective contraction of muscle. That is just one way in which the heart may stop. The usual way in babies is that the heart just simply slows and then stops. But in a proportion which, in this particular group of patients, was somewhere around 25 per cent, ventricular fibrillation may be the mechanism.



E4

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2 MR. SCOTT: Q. Well, I want to
3 take you back. You told us that heart stoppage,
4 the final event, that could be caused two ways,
5 typically.

6 A. Yes.

7 Q. One, by the heart slowing
8 down and stopping or simply stopping.

9 A. Yes.

10 Q. It doesn't necessarily have
11 to slow down, does it? It may just stop.

12 A. Yes. But most times, it
13 does slow down.

14 Q. All right.

15 THE COMMISSIONER: I think that it
16 would have to slow down even if it is almost immeasur-
17 able, but it would have to slow down somewhat before
18 it stopped, I would think, does it not? I'm not sure
19 that --

20 MR. SCOTT: Why?

21 THE COMMISSIONER: Well, I don't
22 see how, when you're going and you stop, you don't
23 slow down.

24 MR. SCOTT: Well, a pump that is
25 pumping at a given rate can stop instantly, it simply
stops the next pump; it doesn't have the next pump.



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E5 2 But no matter.

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4 THE COMMISSIONER: Well, I suppose
5 it can. I don't imagine though that is what
6 happened, but perhaps I'm wrong. But that isn't
7 usual, is it?

8
9 THE WITNESS: Most often there is
10 bradycardia or slowing first.

11 MR. SCOTT: All right.

12 THE COMMISSIONER: Yes.

13 MR. SCOTT: Q. The second method
14 that you were telling us about is fibrillation, the
15 circumstance in which the contractions continue to
16 occur but are chaotic. It isn't necessarily that they
17 are weaker, is it, though they may be?

18 A. They are weaker and they are
19 completely disorganized so that there is no uniformity
20 to the function at all.

21 Q. All right. And the result
22 of fibrillation, that chaos in the pumping, is in-
23 sufficient blood and therefore oxygen doesn't get
24 out to the system?

25 A. There is no blood discharged
to the system.

Q. Yes. Well now, I asked you -
we were going through Mr. Lamek's list - is ventricular



Rowe
ex. (Scott)

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E6

2 fibrillation found from time to time in cases of heart
3 failure which causes cardiac arrest?

4 A. Yes.

5 Q. Yes. Now, how about
6 arrhythmia, is it found from time to time in heart
7 failure which gives rise to cardiac -- the ultimate
8 stoppage of the heart?

9 A. Yes.

10 Q. What about shallow respira-
11 tion?

12 A. That, too.

13 Q. Yes. So, all those factors
14 which Mr. Lamek emphasized, if I read his examination
15 right, can be found in cases of heart failure, which
16 is, on our list, the first cause of cardiac arrest?

17 A. Yes.

18 Q. Yes.

19 Well now, let's come to a second
20 cause of cardiac arrest - hypoxia.

21 I wonder if you could tell the
22 Commissioner what hypoxia is.

23 A. Hypoxia is a lack of oxygen.
24 It is not complete absence of oxygen but it is a
25 considerable and important lack of oxygen available to
the systems of the body.



Rowe
ex. (Scott)

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E7

2 Q. May that lack of oxygen
3 occur in either the heart muscle or the brain?

4 A. Yes. In fact, if anybody is
5 hypoxic, it affects all organs but it particularly
6 affects those which need to have oxygen particularly
7 strongly for their effective action, like the brain
and the heart.

8

9 Q. And what is the consequence
of this lack of oxygen in the brain or heart muscle?

10

11 A. Well, the lack of heart
oxygen affects the cellular function of the organ.
12 It does not allow the organ to produce energy in the
usual way; it interferes with the membranes of the
13 organ and of the cells of the organ and, so, has that
sort of effect.

14

15 Q. Does it have any affect on
the contractions or the pumping that the heart does?

16

17 A. Yes, it can do. Now, the
heart is fairly tolerant of hypoxia in babies.

18

Q. Yes.

19

20 A. So that you can have low
oxygen, a lowered oxygen in the system without
21 necessarily causing any immediate problem in terms of
22 slowing of the heart. But when other events are added
23 to it, such as acidosis or anything that would increase
24 the metabolic demands - the actual requirements of the

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Rowe
ex. (Scott)

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E8

2 body, such as fever or irritability or something
3 like that - that can tip the scale and, then, the
4 effect of hypoxia can completely slide over the
5 scale.

6

Q. I just want to be sure I
understand its effect. If you have hypoxia at an
extreme level --

7

A. Yes.

8

Q. -- which is the shortage of
9 oxygen in the brain or in the heart muscle, do I
10 understand you to say that what that does is reduce
11 the effectiveness of the heart muscle as a muscle?

12

A. It can.

13

Q. Yes. And what effect does
14 that have in an extreme case on contractions?

15

A. Well, it will reduce con-
16 tractions.

17

Q. Yes. To what point?

18

A. To the point where it can
stop.

19

Q. All right.

20

Now, I take it that hypoxia can
stop heart contractions even in the case of a per-
fectly normal heart.

21

A. Yes. We're not quite sure,
in that situation, whether that effect is direct on the

22



1
E9 2 heart muscle or whether it is a brain effect.

3 Q. All right. Well, let me
4 take the case of a baby in the hospital who has a
5 deformed heart. That baby is at risk - I am not
6 asking you to quantify the risk - is at risk
7 theoretically because of the first cause of death,
8 heart failure, stress created by a deformed heart,
9 but may also theoretically be at risk if hypoxia sets
in?

10 A. Yes.

11 Q. And in both cases the terminal
12 event will be a cardiac arrest or the stopping of
13 the heart?

14 A. It could be.

15 Q. Yes. One will -- for one,
16 the first, the cause will be the abnormality of the
17 heart; in the second theoretical case, the cause will
be hypoxia?

18 A. Yes.

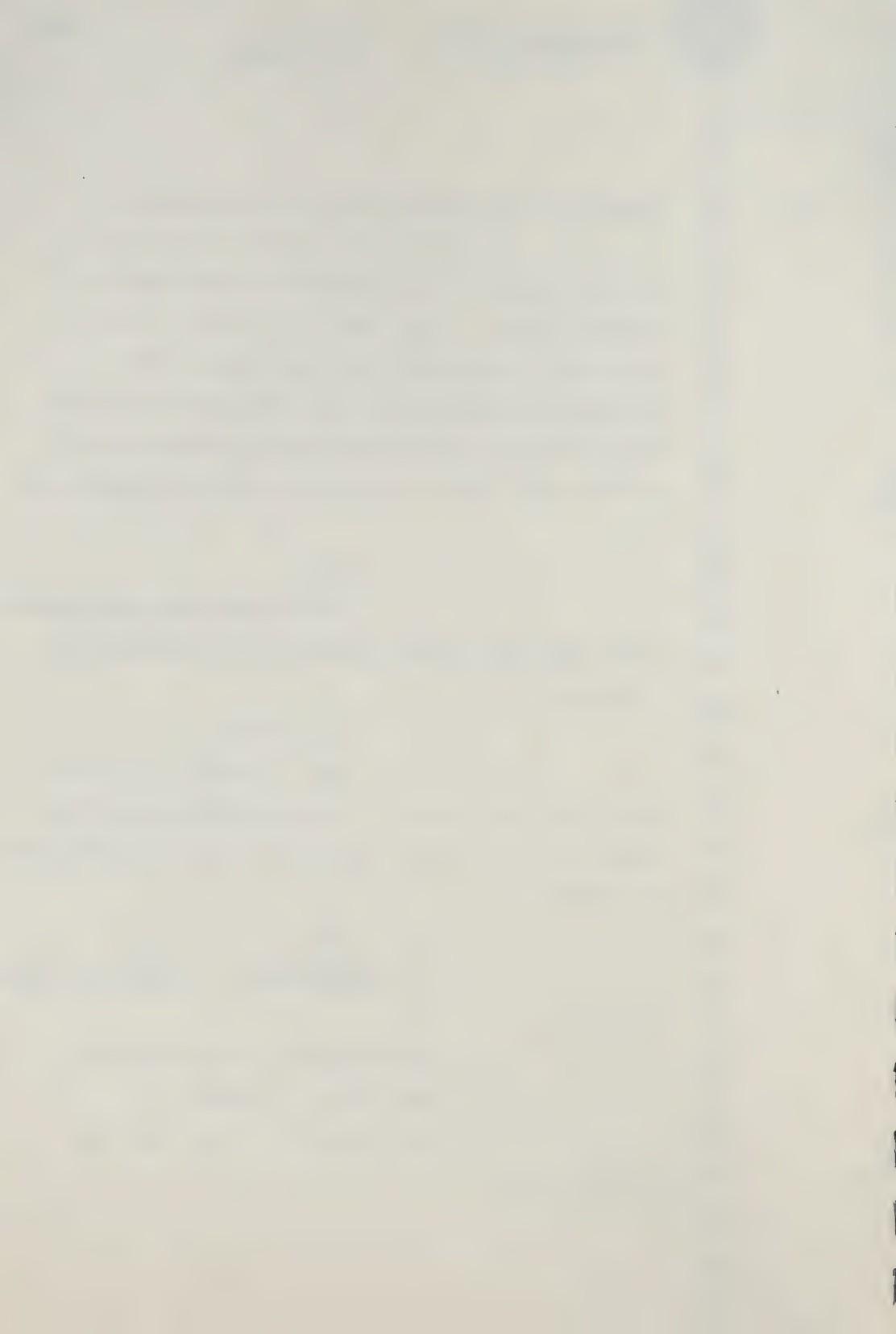
19 THE COMMISSIONER: It could be both,
20 though?

21 THE WITNESS: It could be both.

22 MR. SCOTT: Exactly.

23 THE WITNESS: It can have many
24 combinations.

25





Rowe
ex. (Scott)

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E10

2 Q. The more options there are
3 for causing the stoppage of the heart, Dr. Rowe, I
4 suggest to you, the more difficult it is to determine
5 what caused the heart to stop?

6 A. Yes, that's true.

7 Q. Because the range of the
8 possibilities gets greater, am I right?

9 A. Yes.

10 Q. All right.

11 Now, let's talk about hypoxia. Were
12 there babies in this ward who you think may have
13 suffered from hypoxia as they approached their deaths?

14 A. Yes.

15 Q. All right. Now, I am going
16 to ask you at the end to make, when you have some
17 free time, a list - I haven't asked you to do this -
18 of the babies that you think would be in that
19 category.

20 THE COMMISSIONER: But before we
21 get on with that, I take it that you can suffer from
22 hypoxia without any disease of the heart; is that
23 possible?

24 THE WITNESS: Yes, you can. That is
25 usually from disease of the lung.

MR. SCOTT: Q. Well now, let me



- 1
2 Ell just go at hypoxia again and take Mr. Lamek's list.
3
4 If you had a death that was being
5 caused by hypoxia where there was no structural defect;
6 in other words, I'm talking theoretically - take the
7 pure hypoxia death.
8 Are you with me so far?
9 A. Yes.
10 Q. Might that be accompanied by
11 bradycardia?
12 A. It is.
13 Q. It is?
14 A. Yes.
15 Q. Might it be accompanied by
16 vomiting?
17 A. Yes.
18 Q. Might it be accompanied by
19 sudden deterioration or onset?
20 A. Yes.
21 Q. Might it be accompanied by
22 ventricular fibrillation?
23 A. Yes.
24 Q. Might it be accompanied by
25 arrhythmia?



- 1
2 E12 shallow respiration?
3 A. Yes.
4 Q. Yes. Might it be accompanied
5 by seizures?
6 A. Yes.
7 Q. So, when you have hypoxia,
8 you may exhibit all or some or any combination of
9 those symptoms as you approach the climactic moment
of death?
10 A. That's true.
11 Q. All right.
12 Well now, that's hypoxia, and I will
13 be asking you in the end for a list, but are you aware
14 now of any babies in the epidemic period in 4A and 4B
15 who had both heart abnormalities and were, therefore,
16 susceptible to heart failure and the symptoms and
signs of hypoxia?
17 A. Yes.
18 Q. Now, the third, are you
19 familiar with the term "sepsis"?
20 A. Sepsis, yes.
21 Q. Now, is sepsis itself a
potential independent cause of heart stoppage?
22 A. Yes.
23 Q. Yes. Can it exist without



Rowe
ex. (Scott)

1
E13 2 any malformation in the heart at all?

3 A. Yes, it can.

4 Q. Yes. Now, would you tell
5 the Commissioner and me what sepsis is.

6 A. Well, sepsis is a term that
7 is used, that I suppose would best say that an
individual is septic; that is, that he has a --

8 Q. At the Municipal Board, that
9 has a specific connotation; so, I would be careful
10 about using it.

11 A. He has a bloodstream infection.

12 Q. Is that a bacterial infection?

13 A. With bacteria, usually, yes.

14 Q. And when a baby has that
bloodstream infection --

15 THE COMMISSIONER: Is this
16 demonstrated by pus?

17 THE WITNESS: No. It may not be
demonstrated by pus at all.

18 THE COMMISSIONER: How? What?

19 THE WITNESS: The only way that you
can tell -- it may be demonstrated by pus at some
20 stage, but in many babies, particularly where they -
21 and this is the group we are really, I think, looking
22 at mostly, in the patients that are under consideration
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in this hearing, the babies are young and the speed with which sepsis can cause death in the very young babies is extraordinarily rapid; so that there may be no time for pus to appear visibly anywhere. So, the only way that this can be recognized is by the overwhelming nature and rapidity of the illness and the finding of bacteria in the bloodstream.

MR. SCOTT: Q. Well, just to be sure, Dr. Rowe, that I have it, I take it sepsis -- would you call it a disease?

A. Yes, it is a disease.

Q. All right. And it is a disease that might exist entirely apart from any abnormality of the heart?

A. Oh, yes.



1

2 Q. But may exist with an abnor-
3 mality of the heart?

4

A. Yes.

5

Q. But they are unconnected as

6 a matter of theory?

7

A. Yes.

8

Q. And when you have a blood
9 stream infection that is called sepsis, what does
10 that do to make your heart stop?

11

A. Well it produces a toxic
12 effect on the heart, the toxins from the bacteria.

13

Q. In the blood?

14

A. In the blood. It affects the
15 heart function by a direct toxic effect, especially
in sepsis in small babies.

16

Q. Right. And the toxic effect
17 I take it is the infected blood going to the heart
and the infection being transmitted to the heart
18 muscle?

19

A. The toxins from the bacteria,
20 the release of materials from bacteria which is toxic,
21 the cells.

22

Q. Does that affect the heart
23 muscle cells?

24

A. Yes, it affects every cell.

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F2 Q. And what may the result be
if that is uncontrolled?

A. Death.

Q. Well, how is the death caused?

You will have to move with me at kindergarten level.
You say death, when the toxin has travelled to the heart and infected the heart muscle what effect classically might it have on contractions in the heart?

A. The end result is the failure of the muscle to contract, but the method of getting there is that the toxin affects the membranes of the heart, affects the energy arrangements within the heart and so it disrupts the whole biochemical function of the heart.

Q. But is the result in the end that the contractions of the heart stop's?

A. Yes.

Q. And do you then have a heart stoppage or a cardiac arrest?

A. Yes.

Q.. And that cardiac arrest may, in the use of layman's terms, be caused by blood poisoning that has no relation to a defective heart.

A. That is correct.



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F3 Q. And where there cases of that
in the epidemic period in Wards 4A/4B, or cases
that suggested that as a cause?

5 A. There were cases that suggested,
6 I am not sure of the total, but blood stream infection
7 is something that everybody thinks about immediately
8 a baby is seen to be ill because of the fact that
9 unless you act on it right away then you lose the
opportunity to save the baby. There will be, I think,
10 many instances in that series of patients, in whom
11 action was taken by physicians because of a real
12 concern that sepsis might be operating, not always
13 sustained by the subsequent analysis, but they had
14 to act with antibiotics and supportive measures
immediately.

15 Q. So if you have sepsis in a
16 baby with a normal heart, is it dangerous?

17 A. Yes, it is very dangerous.

18 Q. And can lead to death?

19 A. Yes.

20 Q. If you have sepsis in a baby
21 with a grossly deformed heart, is the danger any
22 different?

23 A. It is higher.

24 Q. Why?

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A. Because the heart is already -

if the heart is abnormal and not functioning well, normally because of its malformation, then if you load it with toxic effect you can make things that much worse. On top of that the sepsis produces a huge increase in the metabolic demand on the heart, that is this fever, the heart is beating faster and you put a new load as well as a toxic effect of the bacteria on the heart. So it is a combination that is pretty lethal.

11

12

13

14

Q. So the sepsis not only poisons the heart muscle, but the presence of the sepsis leads the heart to be obligated to contract harder and faster?

15

A. That is correct.

16

Q. To offset the sepsis?

17

A. Yes.

18

Q. So wouldn't the new word, people call that a synergistic effect?

19

A. Yes.

20

Q. I have wanted to use that word for some time.

21

MR. PERCIVAL: How do you spell that?

22

MR. SCOTT: I haven't the faintest idea.

23

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Q. To make it just clearer, if you have a baby with a congenitally abnormal heart who has both hypoxia and sepsis, is the risk of heart stoppage increasing?

6

A. Yes.

7

8

Q. And are you ever able to say with any finality which of the mechanisms was the dominant mechanism to cause death?

9

10

A. It is sometimes very difficult to do that.

11

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Q. Now let us deal with sepsis.

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You have told us that it may occur even when there is a perfectly normal heart, and I take it it does in your experience?

A. Yes.

Q. May it be accompanied by bradycardia?

A. Yes.

Q. May it be accompanied by vomiting?

A. Yes.

Q. May it occur suddenly, or a deterioration occur quickly?

A. Oh, yes. We have had examples in the neonatal period where babies have been admitted



1

2 and while they are being examined, having been thought
3 to be at least only moderately ill, just suddenly die.

4

Q. In moments?

5

A. In moments.

6

7 Q. You see the trouble with this
is that as I approach my final event as we seem to be
8 calling it here, I anticipate that I will be allowed
9 a leisurely period when I will move in and out of
10 hospital suffering from heart failure and be allowed
11 to slide gradually off the precipice, that is how we
12 like to think about these things. Does that happen
in the case of babies?

13

A. It can happen but it is much
more common that they will deteriorate more rapidly
than that.

14

Q. And why is that, I'm thinking
of something else now, but why is that?

15

A. Well, it is a combination of
18 events. There are a whole host of things that occur.
Babies can become acidotic very quickly so that
20 they have the additive effect of acidosis. They can
stop breathing, especially if they are small. I am
22 talking about small babies which is really what we
are talking about in general. If they have had
24 heart disease that has affected them for some weeks

25



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2 or months, then they have usually failed to thrive,
3 so they have fewer energy stores, they are not as able
4 to respond to the demands that are put upon them if
5 they have any additional infection, or a problem of
6 that sort.

7 Q. I have taken you off course
8 here for a moment. I just want to be sure I have it.
9 We have dealt so far with three causes of heart
10 stoppage: heart failure; hypoxia; and sepsis. Do
11 I have it right that all the indicia that may
12 accompany digoxin toxicity may be found in the manner
of babies dying from those totally unrelated disorders?

13 A. Yes.

14 THE COMMISSIONER: I'm sure that is
15 right, but you stopped at the end, half way through
with sepsis.

16 MR. SCOTT: Oh, did I?

17 THE COMMISSIONER: Yes. Does it
18 also include ventricular fibrillation, arrhythmia,
19 shallow respiration and seizures, or does it
encompass those?

20 THE WITNESS: Yes.

21 THE COMMISSIONER: Does it exhibit
22 those things?

23 THE WITNESS: It can. It depends

24

25



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2 upon the age a bit as to what proportion of babies
3 with normal hearts that will have ventricular
4 fibrillation. It is unusual for babies with normal
5 hearts to have ventricular fibrillation as a means
6 of stoppage, but it is possible for them to do that.

7

8 Q. I take it it is not a matter
9 of concern here, because with three exceptions all
10 the babies had abnormal hearts of various degrees
11 of severity?

12

A. Yes.

13

14 Q. Well now, number 4, "Respiratory
15 Illness".

16

17 MR. PERCIVAL: Mr. Commissioner,
18 what about the other three? We talked about
19 fibrillation, what about the other three?

20

21 MR. SCOTT: The other three modes
22 of dying that I referred to ---

23

24 MR. PERCIVAL: I'm talking about
25 the arrhythmia and shallow respirations and the
seizures.

26

27 THE COMMISSIONER: You indicated
28 that those may be evident.

29

30 THE WITNESS: Yes.

31

32 THE COMMISSIONER: As sepsis?

33

34 THE WITNESS: Yes, indeed.

35



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MR. PERCIVAL: Thank you,

3

Mr. Commissioner, I didn't hear it. Thank you.

4

5

MR. SCOTT: Q. I think you have told us that there are cases that suggested the septic problem in this epidemic period?

6

A. Yes.

7

8

9

10

Q. Now, number 4 "Respiratory Illness". Will you tell us what that is and what impact, if any, it may have on contractions in the heart?

11

12

13

A. Well, the respiratory disorders are of a large, and different number of specific types, but the most ---

14

Q. May I interrupt you, Doctor?

15

A. Yes.

16

Q. What do you mean when you say a respiratory disorder?

17

18

A. Anything wrong with the lungs or the airways to the lungs.

19

Q. All right.

20

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A. So you may have disturbances that interfere with not the lung itself but with the way in which air gets to the lungs, obstructs the airway. In a small baby it is very important because small babies have difficulty if they have minor

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obstructions of the nasal passages, they can very
nearly asphyxiate if that happens on occasion,
because they are not very good at breathing through
the mouth.

6

Q. Or blowing their nose?

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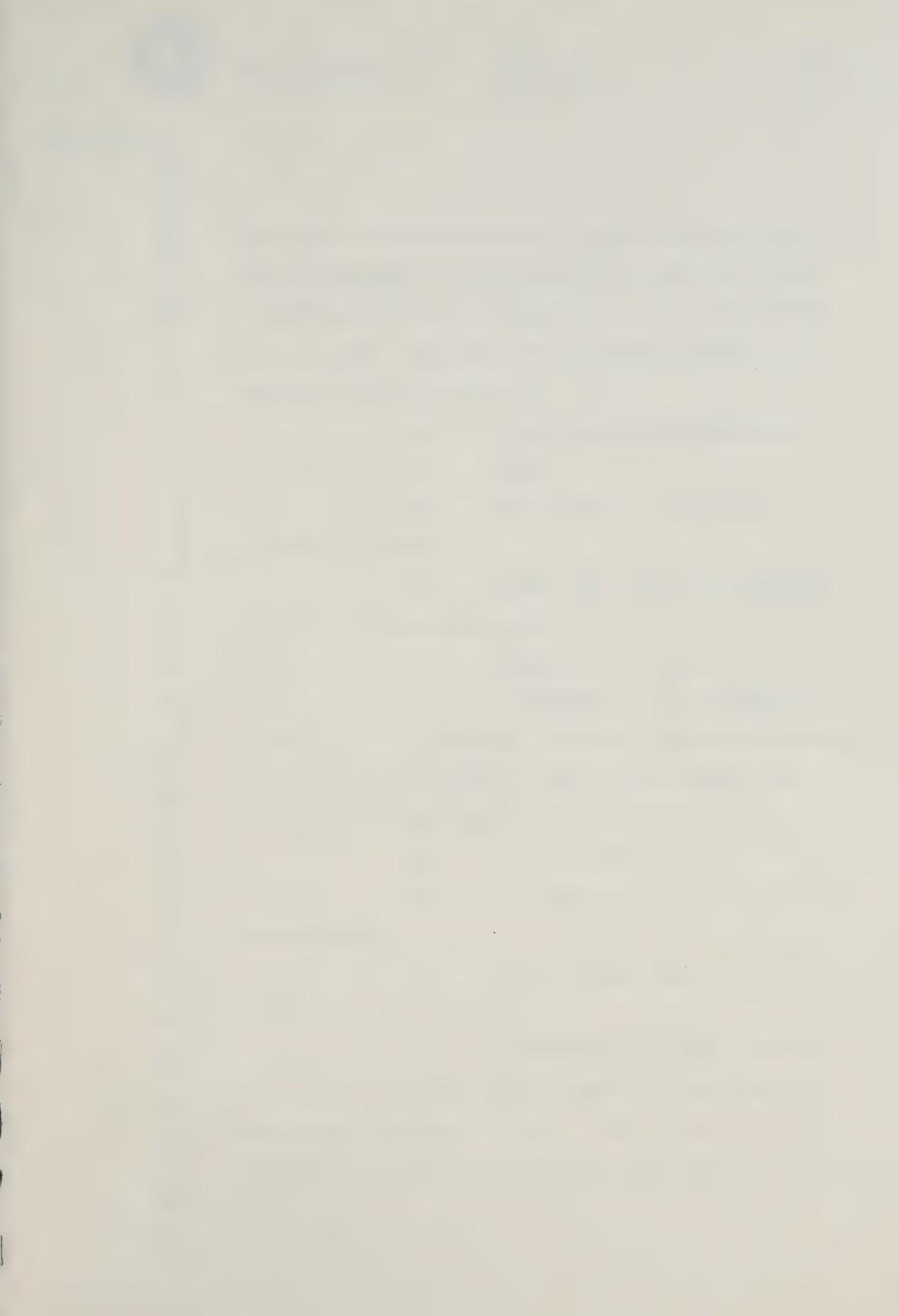
A. That's right. In fact there
are a number of conditions described in young
children where airways obstruction can produce
very severe and fatal consequences. Even having
tonsils that are too big and meet in the middle,
like kissing tonsils, may obstruct the airways in
children to the point where can die.

Q. I take it what that kind of
obstruction does, it is a respiratory ailment or
illness that prevents air from getting to the lungs.

A. Air getting in and carbon
dioxide getting out.

Q. And if that happens what is
the effect on heart contractions?

A. Well, if you have an obstructive
airway like that there is tremendous respiratory
effort, that is this tugging and heaving to try and
get air in and out. That can produce enormous
fluctuations in the - pressures that are inside the
chest. Normally there is a negative pressure inside





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the chest, but when you are struggling to get air in
the effort that can be induced by a baby is of such
a magnitude that you can produce a swing in pressure
that is about the level of your blood pressure, an
enormous swing of pressure.

6

Q. That is airway obstruction?

7

A. Yes.

8

Q. As a kind of respiratory
9 illness are there?

10

11

A. Well, you can have pneumonia,
that is a fairly common one.

12

Q. What is it I mean?

13

14

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A. Pneumonia is an inflammatory

condition of the lungs at the sac level of the alveolar
sacs which is the distal part of the lungs, the
part where the exchange of air is taking place.

Q. Is it viral?

A. It may be viral, or it may
be bacterial.

Q. What is the effect of that
infection?

A. The effect of that infection
is to interfere with the exchange of gases, oxygen
and carbon dioxide. It has a toxic effect if it is -
from the bacteria as well. Those are the main causes.



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Q. Can it lead to a stoppage of
contractions?

3

A. Yes.

4

Q. Of the heart muscle?

5

A. Yes, it can.

6

Q. And cardiac arrest?

7

A. Yes, it can, and especially
this is true in small babies.

8

Q. Now, what about atelectasis?

9

A. Atelectasis is a collapse of
a part, or segment, or part of the lung.

10

THE COMMISSIONER: Can we have that
spelled?

11

THE WITNESS: A-t-e-l-e-c-t-a-s-i-s.

12

THE COMMISSIONER: Yes, thank you.

13

MR. SCOTT: Q. What is atelectasis?

14

A. Actelectasis is a collapse of
a portion, or a total collapse, meaning the whole of
one lung or both lungs.

15

Q. And is that, generally
speaking, a respiratory illness of the type we are
talking about?

16

A. Yes, regardless of respiratory
illness.

17

Q. What is the consequence of
that?

18



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Rowe, ex.
(Scott)

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A. Well, it depends on the severity. It is fairly common in ^many babies who have segmental or collapse of part of a lobe, not to be necessarily seriously affected by that. If it becomes extensive, or if it involves large amounts of smaller segments of lung then it can interfere with the oxygen exchange and carbon dioxide release.

Q. What is lung congestion, is that a respiratory illness?

A. Lung congestion is when the lung is full of blood, and that is usually because of the presence of heart failure. So that in most patients who have heart failure there is a degree of interference with lung function. Sometimes in small babies the actual size of the heart when it is deformed, and malformed and there is heart failure, can be such that it will compress the lung and so cause collapse.



Rowe
ex. (Scott)

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Q. Well, under the general heading respiratory illness, we have dealt with four different kinds of respiratory illness. Are there others as well?

6

7

A. Oh, there are probably others, but that is the major group, I would think.

8

9

Q. All right. And can each of those lead, if severe enough, to stoppage of heart contractions and cardiac arrest?

10

11

12

A. Yes, they can.

Q. And can that occur even in the case of a normal heart?

13

A. Yes.

14

15

Q. Now, add a respiratory illness to a case of an abnormal heart, and what do you get?

16

A. You have an additive effect.

17

18

Q. Yes. And were there cases in this epidemic period where babies died and where there is evidence pointing to respiratory illness --

19

A. Yes.

20

Q. -- as one of the causes?

21

A. Yes, there were.

22

23

Q. Let me take you through the list again. Is death -- that is heart stoppage -- caused by respiratory illness, commonly accompanied by

24

25

If the only time he has seen vent. fib^b where respiratory problems are the major cause of death is one of the patients here [find out which one] is it not apparent that the occurrence of vent. fib^b points away from resp^a problem as cause and to dig in tox^b of which fib^b is known to be a symptom?



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G2

2 bradycardia?

3 A. Yes.

4 Q. May it be accompanied commonly
5 with vomiting?

6 A. Yes.

7 Q. May it be accompanied -- does
8 it exhibit a sudden onset or a sudden deterioration
9 in the patient?

10 A. Yes.

11 Q. May it be accompanied by
ventricular fibrillation?

12 A. I don't know. I haven't seen
13 that, but it may. — *How know if haven't seen?*

14 Q. All right. May it be
15 accompanied -- I only want you to tell us --

16 A. Yes, we do, in fact, have one
17 patient --

18 Q. -- you are quite right, if you
have seen it or have read about it.

19 A. We have one case in this series.
20 I have forgotten one case.

21 Q. This series you are talking
about?

22 A. Yes.

23 Q. May it be accompanied by



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arrythmia?

3

A. Yes.

4

Q. May it be accompanied by
5 shallow respiration?

6

A. Yes.

7

Q. May it be accompanied by
8 seizures?

9

A. Yes.

10

THE COMMISSIONER: At some point,

Mr. Scott, whenever it is convenient...

11

MR. SCOTT: Now.

12

13 THE COMMISSIONER: I'm not too sure
14 how the logistics are going to work out, so we will
15 make it 20 minutes and you can tell me if you don't
16 need that another time.

17

We will take 20 minutes.

16

---Short Recess.

17

18 MR. SCOTT: Mr. Commissioner, if you
19 will permit me, I am going to take another crack at
this chart.

20

21 THE COMMISSIONER: Yes. Just before
22 you do, I want to, just for the comfort and solace of
23 the inner man or woman, we have -- Mr. Diplock of
24 the Municipal Board was concerned about the comfort of
counsel, and they have arranged... There is a lounge

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on the 19th Floor, which has a very simple-minded --
it is where the Assessment people -- the people have
taken over from the County Court in the Assessment
Appeal, where they are there. They are not working
today apparently but they have a lounge. It is
available for Counsel.

There is a combination which is a very
difficult combination to work. If you forget the
combination, you use a credit card and you can get in
very easily. Mr. Lamek says that we can give you
some supplies or something like that and we just hope
that you behave and don't litter the place because
when they do come back and get working again, they
may take objection if there is too much noise. But
they are really trying to make you comfortable. So
if you want to use that for coffee purposes at the
breaks, just do that.

17

18

19

It takes a little time to get up
there. There is the little complication about getting
in, but that is it.

20

MR. LAMEK: Room 1900.

21

THE COMMISSIONER: 1900, yes.

22

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MR. LAMEK: And I suggest not to be
used until tomorrow, and we can get some supplies up
there.

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G5

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THE COMMISSIONER: Yes. All right.

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MR. LAMEK: It is on the other bank
of elevators.

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THE COMMISSIONER: Now, Mr. Scott, do
you want to take over?

7

8

9

MR. SCOTT: Yes. It may be that I
was not clear when explaining one matter on this
chart and that I failed to disclose another matter.

10

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13

First of all, the 5A/4A/4B line in
blue, the ICU line in yellow, the all others line in
purple, the 7G line in brown and the all deaths line
are all geographic lines. That is they are established
by pinpointing the place where the patient died.

14

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THE COMMISSIONER: So that the purple
line is geographic as well on this?

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MR. SCOTT: Yes. Let me just explain:
5A/4A/4B indicates that those patients died in that
ward. The ICU indicates that they died in the ICU.
The 7G indicates that they died in the 7G Neonatal
Ward. The all others is the balance of hospital
deaths that occurred in the hospital.

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So that if you take the 5A blue line,
the ICU yellow line, the 7G Neonatal line and the
all others purple line, you would get the total of
all deaths in the hospital.



G6

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THE COMMISSIONER: Then the purple
line is also geographic?

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MR. SCOTT: Yes.

5

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THE COMMISSIONER: And it is only the
red line that is diagnostic?

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MR. SCOTT: Only the red line is
diagnostic, and I think I should tell you, Mr.
Commissioner, what our evidence will be as to how that
is prepared.

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It is based upon the diagnosis as it
appears in the hospital's records under what is called
the Annual Death Index Print-out, and the criteria
for inclusion are all patients who died on cardiac
wards, so the cardiac line includes all persons who
died on cardiac wards, all congenital heart and
circulatory malformations, except brain, all heart
disease, all heart failure -- technically, so-called --
all heart injuries.

What it excludes are cases of cardiac
arrest because, as we now know, that is the manner of
all deaths.

THE COMMISSIONER: Excludes all cardiac
arrests --

MR. SCOTT: Yes.

THE COMMISSIONER: -- that didn't



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arise by reason of a heart --

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MR. SCOTT: With one exception. It includes cardiac arrests if the patient arrived at the hospital with that diagnosis. That is, if the patient arrived at the hospital in cardiac arrest. It excludes multiple anomalies and we will come to what is meant by that later, where the heart is not mentioned as a factor, and it excludes Down's Syndrome deaths where the heart is not mentioned as a factor.

THE COMMISSIONER: Yes. All right.

EXAMINATION BY MR. SCOTT (CONTINUED)

Q. Now, Dr. Rowe, we had dealt with four categories and I want to take you to the fifth, instability of temperature.

Is this occasionally called, in your evidence, hypothermia?

A. Hypothermia, yes, it is.

Q. Yes.

A. It is one of the manifestations.

Q. And are there patients in the epidemic period who exhibited the symptoms of hypothermia?

A. Yes, there are.

Q. All right. Now, can you tell the Commissioner what instability of temperature is?



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A. Well, that usually means that a patient -- theoretically, it means that a patient may have a temperature that is likely to go from normal to subnormal or above normal, but in practice, the term, at least in young infants, means that they are unable to sustain a normal temperature as well as a healthy infant.

9

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12

Q. All right. Now can I ask you what is the part of the body that controls or creates that -- controls body temperature or creates that condition of instability of temperature?

13

A. It is the brain.

14

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Q. All right. So would it be correct to say that a child who exhibits instability of temperature is disclosing some kind of neurological or brain deficit or disorder?

20

A. It may be. That is often the case. The exact explanation for some babies is not that certain.

21

Q. Is the phenomena easily observed?

22

A. Yes, it is.

23

Q. I suppose, simply by taking the temperature?

24

A. Yes.



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Q. But do I understand you to say
its origin is not in every case clearly understood?

4

A. No.

5

6

Q. Is the brain a candidate for
the originator of this disorder?

7

A. Yes.

8

Q. Now, can you tell us what you
know -- I should ask you more directly -- does
instability of temperature lead, in certain cases,
to a stoppage of contractions of the heart?

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A. I don't know that we know for
sure that that can occur, but in babies who have
unstable temperature there is a tendency for there to
be sudden death so I presume it has got some
relationship.

Q. All right. Can you tell us
anything about the connection between instability of
temperature and a heart stoppage? Why shouldn't the
heart just go on pumping regularly? I'm not talking
about a normal heart. Why shouldn't the heart just
go on pumping regularly as your temperature drops?

A. I think the problem arises
because when your temperature drops the body makes
automatic efforts to raise it again, so it increases
the metabolic rate to bring the temperature back up



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again. Or at least that is the mechanism that is
homeostatic or keeps people the same temperature all
the time.

5

6

Q. Don't confuse me now. Simple
words.

7

A. Simple words. And --

8

Q. Did I interrupt you?

9

A. If you have -- the sort of
baby who has unstable temperature as a small baby and
the attempt to drive up the metabolic rate may be too
much for a baby who has got -- who is small and in
many instances has poor energy reserves.

13

These are babies who are small and

they are not fat like chubby, bigger babies.

14

Q. You have left me with a gap
here. The result of hypothermia is a tendency of a
body to increase a metabolic rate. What, if anything,
does that do with the heart or lungs?

18

A. Well, it places huge demands on
the heart.

20

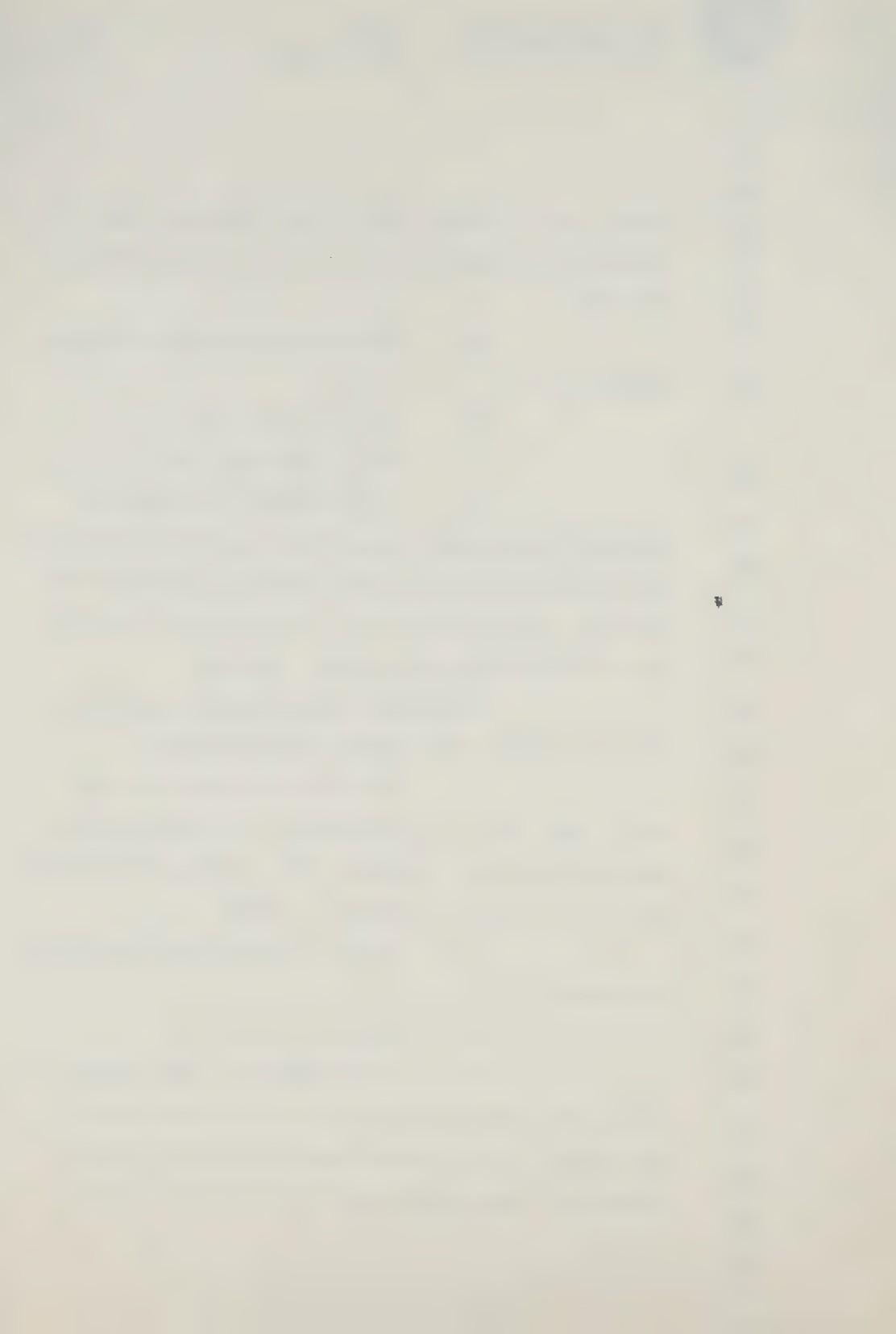
Q. Why?

21

A. The attempt to raise temperature
means that all energy sources are directed towards
the brain, and in the patient who has poor energy
resources, that leaves very little for the rest of

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the systems.

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Q. And what is the result in a
clear case?

5

6

A. Well, that can cause cardiac
arrest.

7

Q. Yes. Now --

8

9

THE COMMISSIONER: I'm sure it can but
I still don't quite know how. How is it effecting the
heart?

10

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14

THE WITNESS: Well, because it has
taken all the energy that is available to try and
stimulate the brain centres for temperature control,
and therefore robs, as it were, the other systems of
the body, particularly the heart.

15

16

17

18

Q. When this got us going,
in response to the increased metabolic rate to the
brain, I take it there is no decrease in the need of
oxygen in other parts of the body if it is to live?

A. No.

19

Q. What does that do to the heart?

20

21

A. Well, that deprives the heart
of the fuel, of the little fuel that is available.

22

23

Q. All right. So the heart is
deprived of oxygen by that mechanism; is that correct?

A. And energy.

24

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Q. Yes.

2

A. Utilization.

3

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Q. That is one thing. Is the
demand on the heart to produce oxygen or to pump
increased?

6

A. Yes, it is.

7

Q. So do those two things occur --

8

A. Yes.

9

Q. In classic hypothermia?

10

A. Yes.

11

12

Q. And may that lead to heart
stoppage?

13

A. Yes, it may. It doesn't make
it always do it, but it may.

14

15

Q. And that can occur, I take it,
in babies with perfectly normal hearts?

16

17

A. Yes, it can.

18

Q. You have seen that?

19

A. Yes. It is a common disorder
in neonatal units.

20

21

22

23

MR. PERCIVAL: Mr. Commissioner, I am
having difficulty with the use of the word hypo or
hyperthermia, and I'm wondering whether or not there
is a distinction between the two.

24

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THE COMMISSIONER: One is high and one



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is low.

3

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MR. PERCIVAL: That is right, and I am
wondering whether we are talking about low temperature
up to this point in time. I am sorry.

6

7

THE COMMISSIONER: Hypo is low and
hyper is high.

8

THE WITNESS: That is right.

9

MR. SCOTT: I take it -- I have
caused this confusion, Dr. Rowe, and it won't be the
last confusion I have caused -- but I take it the
mechanics that we are talking about are the same
whether the instability of temperature produces a
high temperature or a low temperature?

14

A. Yes.

15

Q. The demand of the brain for
oxygen

16

A. Yes.

17

Q. -- is created by either of
those conditions?

19

20

A. Yes, it is, but the usual thing
in small babies is hypo, but it may be hyper.

21

22

Q. And what is hypo again, just
so I won't make a mistake again?

23

24

A. Hypc is body temperature less
than normal.

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Q. You have told us this can occur
in the case of a baby with a normal heart, and I
don't want to keep repeating this question, but a
baby with an abnormal heart structure and hypotherm-
ia, what is the incidence of risk to that baby of
cardiac arrest?

8

A. I think it is additive.

9

10

Q. And I take it if you add
respiratory illness on hypoxia or sepsis, what
happens?

11

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A. It's the same, they all have
a contribution.

Q. Yes. Now, a baby who is
dying of hypothermia or instability of temperature,
will that baby on the passage to brain - I'm sorry,
to heart contractions stopping exhibit bradycardia?

A. Yes.

Q. Yes. Will the baby exhibit
vomiting from case to case?

A. I don't know.

Q. You don't know about vomiting
in that case. And when you say you don't know, just so
we are clear, I take it that means you have never seen
it?

A. Well, this condition is seen
more often in the neonatal floor than perhaps in other
parts of the hospital. So, we see it in sickly babies
who are small with congenital heart disease who are
not necessarily neonates. But I don't recall specific
babies with vomiting, I would have to perhaps refer
to the individual charts of those babies that were
affected in this group we're talking about.

Q. Is the onset of arrest or
deterioration a sudden one?

A. Yes, can be.

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Q. Yes. Is it accompanied by ventricular fibrillation?

A. If the baby has congenital heart disease it may be.

Q. Yes. And you've made that observation before that ventricular fibrillation is usually found in babies with congenital heart deformities?

A. Yes. The reason for the difference appears to be that you need a certain critical mass of muscle before fibrillation can develop. There has to be a certain specific mass of muscle. I don't know that there is a figure of how many grams or anything like that, but that's the theory by which the differences are explained between older individuals who have heart stoppage and younger patients. Younger patients tend to have bradycardia more often than older patients. Older patients tend to have ventricular fibrillation.

Q. Can it be accompanied by arrhythmia?

A. Yes.

Q. Can it be accompanied by shallow respiration?

A. Yes, indeed.



1

2 Q. Can it be accompanied by
3 seizures?

4 A. Yes.

5 Q. Well now, I want to take you
6 to another characteristic to which you have referred
7 in your evidence and that is the case of babies with
8 low birth weights. Now, first of all, is there any
9 connection between low birth weight per se and a
stoppage of contractions of the heart?

10 A. Not necessarily.

11 Q. All right. Is it possible?

12 A. Well, if you have a very, very
13 small baby who is just barely on the viable range,
then that may.

14 Q. All right. What do you mean
15 by low birth weight?

16 A. Well, a low birth weight is
17 usually meant to - it has different definitions from
18 different people but most I think would agree that
19 a baby that is under 2500 grams is a low birth weight
baby.

20 Q. All right.

21 A. And there are varying
22 definitions of low birth weight. Some people talk
23 about very low birth weight babies as being under 1500

24

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grams.

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Q. Well, let's talk about low birth weight babies. Is there any effect of low birth weight on the brain?

6

7

8

A. I don't know that I can answer that question specifically but low birth weight babies are more susceptible to brain injury or brain damage.

9

10

Q. Yes.

A. So, I think that the answer

11

would probably have to be yes.

12

Q. Now, what effect does that have on heart stoppage?

13

14

15

A. Well, I think that the baby who has a low birth weight is not necessarily, but it usually is the case that it is immature in its development; that is premature.

16

Q. Yes.

17

18

19

20

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22

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A. Prematurely born. But some

babies have low birth weight for an appropriate age and duration of the pregnancy. So, there's a little difference between those two types of babies, as I understand from the neonatologists, they are the people who really can answer all those questions in more detail. But no doubt that in a large number of low birth weight babies the brain function has certain



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5 2 immaturity.

3 Q. Yes. Those baby lack fat I
4 take it, characteristically?

5 A. They do lack fat, yes.

6 Q. Does that have any impact on
7 the need for oxygen in the baby's system?

8 A. Well, they have very little
9 in the way of energy reserves.

10 Q. And when you say energy reserves,
11 what do you mean?

12 A. I mean the fuel by which the
13 body can function is limited.

14 Q. All right. Does that have
15 any impact on the possibility of heart stoppage?

16 A. Yes, it does.

17 Q. Yes. And how does it work,
18 what's the process?

19 A. Well, the more fuel reserves
20 you have the longer it's possible to go if you are
21 stressed as a baby of that weight. In taking it in
22 its extreme form, the baby who has a good reserve of
23 sugar in the liver and the heart will survive
24 hypoxia for a longer period than a baby who has
25 limited reserves of those substances. I can't give
you all the biochemical associations of that sort,



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6 2 but I think that's fairly definitely accepted.

3 Q. Well, what I'm trying to get
4 at is whether in the case of a very low birth weight
5 baby there is a connection between that birth weight
6 and heart stoppage that may exist irrespective of the
presence or absence of heart defect?

7 A. Yes. We're not quite sure
8 why that is but that has been an observed phenomena
9 that babies of that weight are subject to sudden and
10 unexpected death, even though the heart may be normal.

11 Q. All right. Now, I'm not asking
12 you to try and get the Nobel Prize, but can you just
13 tell us what you think as a matter of speculation the
14 connection between very low birth weight and heart
stoppage may be?

15 A. Well, it may be because of
16 things that occur in the respiratory centre of the
17 brain, that is, the centre that governs the breathing
18 apparatus and if that for some reason goes out of
kilter, then the baby could stop breathing and that
19 could end up in an arrest.

20 Q. Yes. Well now, if that
21 happens - let me put it this way. We were talking
22 about very low birth weight babies and I take it for
23 very low birth weight babies this can occur in the

24

25



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2 absence of any other indicia of defect or illness?

3 A. Yes.

4 Q. But in the next category, low
5 birth weight babies, is this a factor that impacts on
other diseases, defects of disorders that may exist?

6 A. Oh, yes.

7 Q. So that a baby with heart
8 defect who is low birth weight, what happens to the
9 risk of cardiac arrest in that baby?

10 A. I think it's higher and I
11 think more than that if the baby's weight does not
12 increase normally in the first few months, then those
13 babies who may be of even apparently normal birth
14 weight or close to it, there is a tendency for babies
15 with congenital heart disease to have lower birth
16 weights than the average population, but given that
17 the birth weight is within the usual range and the
18 baby doesn't thrive because of the presence of the
heart defect, then that baby in my view begins to
respond more like babies of low birth weight.

19 Q. All right. So, you can in
20 substance, regardless of your birth weight at birth,
21 you may become like a low birth weight baby if you
22 fail to thrive after birth?

23 A. Yes.

24

25



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2 Q. Now, dealing with a case of a
3 baby of very low birth weight with no cardiac defect
4 who is going to die, will not the manner of dying be
5 typically accompanied by bradycardia?

6

A. Yes.

7

THE COMMISSIONER: From what though,
from the low birth weight?

8

MR. SCOTT: From low birth weight.

9

Am I right about that?

10

A. Yes.

11

Q. Will it be accompanied by
vomiting?

12

A. I'm not sure of that.

13

Q. All right. Is the onset of the
disorder or its deterioration likely to be sudden?

15

A. Yes.

16

Q. Is it likely to be accompanied
by ventricular fibrillation?

17

A. I don't think so.

18

Q. All right. Is it likely to
be accompanied by arrhythmia?

20

A. Yes.

21

Q. Is it likely to be accompanied
by shallow respiration?

23

A. Yes.

24

25



1

2 Q. Is it likely to be accompanied
3 by seizures?

4 A. It could be.

5 Q. Yes. And there are cases in
6 the epidemic period of babies for whom low birth
7 weight may be a factor leading to heart stoppage?

9 Which?

7 A. Yes.

8 Q. Well, now I come to the next
9 general heading Conduction. I had difficulty with
10 conduction as the cause of heart stoppage, so, I
11 divided it into four categories when I discussed it
12 with you and perhaps we can deal with them as four
13 categories because they seem to be slightly different.
14 The first is conduction failure type one, electrolyte
15 imbalance. Now, can you tell me what I'm talking
16 about.

17 A. Well, the ---

18 Q. First of all, if I can
19 interrupt you. What are electrolytes?

20 A. Electrolytes are salts that
21 are in the body normally.

22 Q. Yes.

23 A. And the chief ones that are
24 involved are sodium, potassium and chloride.

25 Q. Yes.



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(Scott)

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2 A. But there are others like
3 calcium and magnesium and other substances.

10

4 Q. Are those salts typically
5 found in every cell in the body?

6 A. Yes.

7 Q. And what do we know about the
8 role that those salts play in those cells?

9 A. Well, they are very important
10 in cell behaviour because there is a different
11 distribution of different substances like potassium
12 and sodium which have different concentrations inside
13 and outside cells. This balance of these ions and
14 salts is critically important to the general behaviour
15 of cells in the body.

16 Q. So, if I can stop you there
17 just to see if I understand it. These salts are
18 found in varying proportions in every cell?

19 A. Yes.

20 Q. In the body?

21 A. Yes.

22 Q. And they are in some way
23 critical to the operation of the body's systems?

24 A. Yes.

25 Q. Now, can you tell me how they
are critical?



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A. Well, you know, I am not a
biochemist.

4

Q. In sentences of 25 words or
less.

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A. I'm not a biochemist and it's a long time since I did my second year of medical school but I think that the important thing is that there are relatively constant relationships of the salts and these metals and these substances like potassium and calcium and magnesium and so on inside on the membranes of cells and inside the cells and outside in fluids surrounding the cell and it is essential that they are concerned with membrane activity and there is movement of these substances across the membranes under certain conditions. If they are disturbed in any way, if the relationships and concentrations are disturbed in any way, that can vitally affect the function and the performance of any particular cell.

19

Q. All right, including the heart?

20

A. Including the muscles of the heart or the nerves of the heart.

21

Q. All right. Now, you've told us that that can happen if there is - how would you describe it?

24

25



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2 A. A disturbance.

12

3 Q. A disturbance in their ---

4 A. In their concentrations.

5 Q. In their concentrations. Now,

6 is that what is sometimes called an electrolyte
imbalance?

7 A. Yes.

8 Q. Now, when there is an electro-
9 lyte imbalance now, you've told us that that can affect
10 the operation of any cells in the body?

11 A. Yes.

12 Q. And of course the heart is made
13 up of cells?

14 A. Yes.

15 Q. How does that imbalance, in
16 words of one syllable, affect the heart, or how can
17 it affect the heart?

18 A. Well, it depends on which is
19 the predominant imbalance. You can have an imbalance
20 that affects one of these substances very much more
21 than another; for example potassium.

22 Q. Yes.

23 A. And if you have a loss of
24 potassium from the body for one reason or another,
25 usually from things like diarrhea and vomiting,



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A. Yes, it can. For example,
if a baby has gastroenteritis, it can very quickly
and very rapidly get into a state of potassium loss
in the body that would do just that.

4

5

Q. So, does that mean that that
imbalance of one of these electrolytes may lead to
heart stoppage?

6

7

8

A. Yes.

9

Q. And cardiac arrest?

10

11

A. Could do. Now, hopefully,
you get to babies before they do that, but it can do.

12

13

Q. Now, you have told us in a
sense how this imbalance can be caused, and I take it,
just to summarize, it can be caused by a diuretic?

14

A. Yes.

15

Q. Diarrhea?

16

A. Yes.

17

Q. How about low intake of water?

18

19

A. Or inadequate intake of
potassium in the diet. I am talking about potassium.

20

Q. Yes.

21

A. You are talking about every-
thing?

22

23

Q. I am talking about the other
two; sodium and chloride, as well.

24

25



- 1
- I2 2 A. Well, you know, there are
- 3 different things that can increase or decrease those.
- 4 Q. Tell us if I am covering the
- 5 things.
- 6 A. Yes, you are.
- 7 Q. A low intake of water.
- 8 A. Yes.
- 9 Q. Low or inadequate feeding.
- 10 A. Yes.
- 11 Q. Vomiting.
- 12 A. Yes.
- 13 Q. Anything else that can
- 14 destroy the balance.
- 15 A. Well, if you are talking about
- 16 a loss of the substance?
- 17 Q. Yes, I'm talking about that.
- 18 A. You may have conditions in
- 19 which the kidney is abnormal, is functioning abnormally.
- 20 Kidney diseases can produce a lot of disturbance of
- 21 these substances. There are a whole host of diseases
- 22 that can either cause an excessive loss of these
- 23 substances or can retain them in the body to an
- 24 excessive level.
- 25 Q. And is this electrolyte
- failure, type no. 1, which we have been talking about,



14

1 characterized by vomiting?

2 A. Yes.

3 Q. Will it be marked by a
4 sudden deterioration or onset?
5

6 A. Yes.

7 Q. Will there be ventricular
8 fibrillation?

9 A. There may be, especially if
the baby has congenital heart disease.

10 Q. Will there be arrhythmia?

11 A. Yes.

12 Q. Will there be shallow respira-
13 tion?

14 A. Yes. And I emphasize that
would be in the case of fairly severe derangement.

15 Q. Would there be seizure?

16 A. There may be.

17 Q. And are there cases in this
18 ward where there is evidence pointing to this kind of
19 difficulty?

20 A. Yes.

21 Q. Now, let me come to conduction
failure type no. 2, which is the eighth cause of death.

22 Are you familiar with a conduction
23 failure that results from the introduction of an

24

25



15 2 extraneous substance?

3 A. Yes.

4 Q. Can you tell us -- can you
5 give us a list of some of the extraneous substances
6 which may be introduced and, thereby, cause a conduction
7 failure type 2? How about digoxin?

8 A. Digoxin, I think we can say
9 is a major contender.

10 Q. Anything else?

11 A. Potassium can do the same.

12 Q. Yes.

13 A. An excess of calcium can do
14 the same.

15 Q. How about quinidine?

16 A. Quinidine, propanolol.

17 Q. How do you pronounce that?

18 I haven't got that right yet.

19 A. Propanolol, p-r-o-p-a-n-o-l-o-l.

20 Q. I take it that these are
21 all natural substances, or therapeutic substances, which
22 may get introduced to the body but which may cause
23 electrolyte imbalance?

24 A. Yes. Well, I am not sure
25 about propanolol and quinidine.

Q. I'm sorry, conduction failure.



16

1 A. Yes, conduction failure.

2 Q. And will you tell the
3 Commissioner - and, by the by, me - how it happens
4 that this may relate to, and cause, a heart stoppage?

5 A. Well, if you look at, say,
6 potassium--I will have to take one or two individually.
7

8 Q. Yes.

9 A. The effect of potassium, when
10 one gives that to an individual, will be to change the
11 speed of conduction of the electrical impulses in the
12 cardiac conduction system. So, the complexes,
13 instead of being, on the electrocardiogram, nice,
14 narrow blips and so on, will broaden out and take a
15 longer period to conduct, and there is sort of a rough
16 relationship between the levels of potassium in the
17 blood and the degree of electrocardiographic disturbance,
18 and it can progress from that to complete heart block
19 and so on.

20 Q. What do you mean by "heart
21 block"?

22 A. Where the impulses at the top
23 chambers are not transmitted through to the bottom
24 chambers, which then begin to start their own rhythm
25 at a slower rate.

Q. Now, leaving aside the reason



1

17 2 why the substance is introduced to the body, can these
3 substances, when introduced, cause a conduction failure
4 of the type you have described?

5 A. Yes.

6 Q. And can that conduction
7 failure lead to heart stoppage?

8 A. Yes.

9 Q. Can that occur in the case
of a perfectly normal heart?

10 A. Yes, it can.

11 Q. Are you familiar with that
12 in the literature and in the clinic?

13 A. Yes.

14 Q. Now, when that happens, when
15 death is going to occur from conduction failure no. 2,
16 will it be accompanied by bradycardia?

17 A. Yes.

18 Q. Will it be accompanied by
vomiting?

19 A. It may be. I am not sure of
the exact proportion in that.

20 Q. Will it be accompanied by,
21 or will it be marked by a sudden onset or sudden
22 deterioration?

23 A. It could be.



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1 heart surgery can produce disturbances in the heart
2 rate and conduction. An obvious one, and one that
3 we sort of anticipate today will be relatively in-
4 frequent, is when the main cardiac nerve near the
5 atrial ventricular node, the portion of the conduction
6 system that passes down to supply the two pumping
7 chambers, when that is interfered with by repair of
8 a ventricular septal defect or some such arrangement
9 in the ventricles. In placing a patch to close a
10 hole, the surgeon has to put stitches around the
11 margins of the patch, and that scenario is very close
12 to where this nerve is - you can't see the nerve; it
13 looks the same as heart muscle and, so, originally, it
14 was a very difficult thing to avoid but, nowadays,
15 surgeons can - except in more complex conditions -
16 make a very good attempt to avoid that. If the
17 stitching involves the nerve or the blood supply to
that nerve, then it can interfere with the conduction.

18 THE COMMISSIONER: What is the
19 name of this nerve again?

20 THE WITNESS: Well, this is the main,
perhaps we should call it the bundle of HIS.

22 THE COMMISSIONER: I wonder if we
could go back to Exhibit 41A.

23 MR. SCOTT: Yes. Is that the big
24 chart?

25



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2 THE COMMISSIONER: It is the big
3 chart, I think, yes.

4

5

THE WITNESS: It might be helpful
to have that.

6

MR. SCOTT: Is that it there?

7

8

THE COMMISSIONER: Yes. That is
something I have. I think it would be better if we could
find the big one.

9

10 MR. SCOTT: Mr. Registrar, which
is the exhibit, the small one or the big one.

11

12 THE COMMISSIONER: It is Exhibit 41A
that we are looking for.

13

14 MR. SCOTT: We can't seem to find it.
Perhaps Mr. Lamek can help us. It is the Pacsai chart.

15

16 THE COMMISSIONER: No. 41A was
one that -- The Pacsai chart will be just as good.

17

MR. SCOTT: Do you know where it is?

18

19 MR. LAMEK: No. If it is not in
that bundle, I don't. It is one of the very first
drawings of the normal heart.

20

21 MR. SCOTT: We have it, Mr.
Commissioner.

22

23 Q. Now, you were talking about
conduction failure type 3, caused by surgery --

24

25 THE COMMISSIONER: Just a moment. I



1
2 want to make sure we know what exhibit we are talking
3 about.

4 MS. CRONK: Exhibit 108, Mr.
5 Commissioner.

6 THE COMMISSIONER: Yes. All right.

7 THE WITNESS: We are not talking
about, Mr. Commissioner, either of those individuals.

8 THE COMMISSIONER: No. I understand
9 that.

10 THE WITNESS: This is just to refresh
11 our memories about the conduction system.

12 There is the sinoatrial node,
13 up in the top portion of the right atrial or right
14 orifice and, from there, there are impulses sent
15 across the top chambers of the heart that then
16 concentrate at the atrial ventricular node. From
17 that point, there is a main bundle of HIS; then the
18 HIS, and that is spelled H-I-S, which is a
19 major nerve structure that looks like muscle but is a
20 specialized muscle, and passes from that AV node
21 until it branches at this point, at the upper
22 part of the ventricular septum into two main branches;
23 the right bundle and the left bundle, and these
24 bundles are the sort of telephone wires, as it were,
for impulses, electrical impulses, to travel down to the
muscles of the pumping chambers.

25



J/EMT/ak

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So that in a repair of a ventricular septal defect in this region here where the patch has to be placed, there is a specific risk that in the course of the repair the nerve may be caught with the suturing of the patch.

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Now it doesn't happen very often now, but at the beginning of this surgery about a quarter of all patients have that problem. Now because surgeons know where that thing goes, they can avoid it in most instances, but there will still be a theoretic risk and there is a potential for that development.

THE COMMISSIONER: But the nerve,

to distinguish it from other nerves, what would you call it?

THE WITNESS: Well, it is not really a nerve in the general appearance of a nerve anywhere else. It is really a specialized muscle. You can't tell where it is if you look at the heart.

You can only know that from examination of the hearts of children who have not survived and of hearts that were belonging to individuals who died before heart surgery became available.

THE COMMISSIONER: I take it if you cut it then all electricity ceases.



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THE WITNESS: At that point.

3

THE COMMISSIONER: Yes.

4

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THE WITNESS: It is like you produce complete heart block. It is just as though at that point you have completely severed the situation. It produces a picture which is like congenital heart block, but it is induced by the surgeon.

The more complicated the defect, the more difficult it is to be sure where that passage is because sometimes it is in front of the hole and sometimes it is behind the hole, and it takes a lot of specialized electrical testing, like an electrician, at the time of operation nowadays to be sure where that is.

In any event even interference with the blood supply from the node by the stitching can cause abnormality of function of that conduction system. So that is one way.

If that happens there is a high probability that that patient would die. It is not well tolerated and usually those patients have to have a pacemaker inserted very quickly.

Any operation on the top chambers of the heart can interfere with the sinus node. And the sinus node is the real pacemaker of the heart

25



1

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under ordinary circumstances. So if you interfere
with that you are left with pacemakers that develop
lower down: either at the AV node here (indicating)
or at various places in the ventricular myocardium
or muscle. And that is quite a serious condition too.

If it occurs, not because people put a
stitch necessarily through the node because they
usually don't go at that point, but it most often
seems to occur by any extensive surgery that is
necessary on the upper chambers can interfere with
the blood supply to the node. So you can have
disturbances of rhythm from the technique of inter-
ference with the blood supply or by direct damage
to the node.

MR. SCOTT: Q. Just so that I will
get some perspective how big is the heart of a baby?
That is a one month old baby?

17

A. Oh, about 28 to 30 grams.

18

Q. That doesn't mean anything to
me.

20

THE COMMISSIONER: About the size of
the baby's fist is what we have heard before.

22

MR. SCOTT: Q. About the size of a
plum.

23

THE COMMISSIONER: And a grown person's

24

25



1

2

about the size of Mr. Lamek's fist.

3

MR. SCOTT: There is not room in
here for that.

5

6

7

THE COMMISSIONER: About the size
of the baby's fist, would that be fair, for the whole
heart?

8

9

10

THE WITNESS: Yes, that would be
fair unless the heart is malformed, in which case
it might be much bigger because it is thickened out
and got stretched a bit.

11

12

13

MR. SCOTT: Q. Do these difficulties
occur in the case only of septum repairs or any
other heart surgery as well?

14

15

16

A. They can occur in any form of
heart surgery because one or other - any form of
intracardiac operation. Operations outside the heart
will not necessarily produce that.

17

18

19

Q. How do you learn that it has
occurred? Do you learn because the baby died and you
do an autopsy?

20

21

22

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A. No, because if you get a
disturbance of rhythm then it will become apparent
on the clinical examination and the monitoring,
especially since these children, postoperative,
would go to the Intensive Care room where there



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would be - Intensive Care area where they would have
3 very close monitoring, electrocardiography.

4

5 Q. Are there any babies in the

epidemic period for whom this might be a consideration?

6

A. I can't recall just offhand,

7 but I could look into that.

8

Q. I am going to ask you to
divide the babies up with regard to these 14 cases.

9

In your spare time.

10

11

MR. PERCIVAL: Mr. Commissioner,
that question was not put on the No. 8 one as well.
I didn't know whether that was an oversight on
13 Mr. Scott's part.

14

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MR. SCOTT: Q. The No. 8 is

digoxin, quinidine, propanolol, other babies for
whom those possibilities are a consideration in this
epidemic period.

A. Oh, yes.

MR. SCOTT: I presume, Mr. Percival,
that was in the nature of a debating point, was it?

MR. PERCIVAL: Oh, no, I just
thought it was an oversight.

MR. SCOTT: No.

MR. PERCIVAL: I am trying to be
helpful.



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THE COMMISSIONER: I can do the same
thing but you perhaps are not finished with this
operating...

5

6

MR. SCOTT: Had you a question,
sir?

7

8

9

THE COMMISSIONER: No. The only
thing is that in every case you have asked, and
certainly you must conduct your case as you want to,
but whether the symptoms ---

10

11

MR. SCOTT: I haven't come to that
yet.

12

13

THE COMMISSIONER: No. I just
want to make sure that you did.

14

15

MR. SCOTT: I won't take as long as
Mr. Lamek went about it.

16

MR. LAMEK: That is a very cheap
shot. Not worthy of you.

17

18

MR. SOPINKA: You should have said
you needed a lot of help.

19

20

MR. SCOTT: This is just the first
morning, Mr. Commissioner.

21

22

23

24

25

Q. If death occurs from conduction
failure, Type No. 3, Dr. Rowe, I take it that that
conduction failure would be attributable to some
defect in the heart because you wouldn't have operated



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on a normal heart.

3

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A. No. That is correct. It is
possible for people as we have seen to have congenital
heart block.

6

Q. Yes.

7 A. One of the patients I believe
8 was a patient who had congenital heart block, so you
9 can be born with that block. There are patients who
10 developed what we call sick sinus which means that
11 the sinus node just doesn't function well who don't
have any other disease of the heart system.

12

13

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Q. Let me see if I understand
then. The conduction failure, the Type 3 that we
are talking about, you illustrated by a reference
to how the failure might be created by a surgical
incident?

A. Yes.

Q. Are you telling us that it
can also exist in the congenital heart?

A. Yes, it can.

Q. And you think there may be
a baby who exhibited that in this case?

A. I am not sure again exactly
of the numbers here.

Q. All right. How about Bruce



1

2

J8 Floryn, for example?

3

A. Yes.

4

Q. Did he exhibit it?

5

A. Yes.

6

Q. All right. We will get at
our list later.

7

I suggested to you that this condition
is not going to occur in the case of a normally
arranged heart. Is that correct? Because it is
itself a defect of the very heart of the child, or
am I wrong about that?

12

A. No, I think - I am not quite
sure of your question.

14

THE COMMISSIONER: Remember we
started off this was an operation.

16

MR. SCOTT: Yes.

17

THE COMMISSIONER: So it is in the
operation, and I thought it was ---

18

MR. SCOTT: No, Dr. Rowe has gone
on to say that an operation is one way it may be
caused. It may exist congenitally. That is there
may be - it is not natural but there may be at birth
a block.

22

THE WITNESS: Yes, or it may be
acquired.

24

25



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MR. SCOTT: Q. How can it be
acquired?

4

5

6

7

A. It probably has its origins
farther back than that but you may for the first time
see somebody who has had heart block that develops
spontaneously at, say, four or five days of age.

8

9

Q. So heart block is the
description of this phenomenon, is it?

10

11

A. Yes.

Q. Do I have it right then that
heart block can onset unexpectedly?

12

A. Yes.

13

Q. Be congenital?

14

A. Yes.

15

Q. Or be caused by surgery?

16

A. That is correct.

17

18

Q. And if that happens what is
the result in relation to the contraction of the
heart?

19

20

21

22

23

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A. Well, the heart can contract
fairly well in a patient who has heart block, complete
heart block that occurs spontaneously without any
underlying disease. Then what happens is that the
contraction of the heart is a bit like a mile runner
or something, some other distance in the new system.



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The heart is slow and the heart enlarges to cope with
a need to deliver the same amount of blood per
minute, but at a slower beat.

5

Q. Yes.

6

7

A. And it contracts fairly
effectively.

8

The problem is that in heart block
that is either surgically created or that is - and
in many patients at least with congenital heart block
the rate is so slow that there is an opportunity for
other pacemakers to start in the heart muscle and so
you get ectopic rhythms or irregularity as well as
slowness.

14

15

16

17

Q. And what does that slowness
plus the irregularity created by other pacemakers
lead to?

A. Well, you can die suddenly

with that.

18

Q. Well, when you die suddenly --

19

A. Ventricular fibrillation.

20

21

Q. Are we talking then about a
heart stoppage?

A. Yes, indeed.

22

23

Q. And would that be accompanied
by bradycardia?

24

25



1

2

A. Yes.

3

4

answered it.

5

A. Yes.

6

7

Q. Would it be accompanied by
vomiting?

8

A. It might.

9

Q. Would it onset suddenly or
would the patient deteriorate suddenly or unexpectedly?

10

A. Could do.

11

Q. Would it be and perhaps you
have answered this, accompanied by fibrillation?

12

A. Yes. Not always but it may be.

13

Q. Would it be accompanied by
arrhythmia.

14

A. Yes.

15

Q. Would it be accompanied by
shallow respiration?

16

A. It may be.

17

Q. Would it be accompanied by
seizure?

18

A. Could be, too.

19

Q. No. 10, conduction failure
Type 4, viral infection of the heart. Are you familiar
with that expression?

20

21

22

23

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A. Yes.

3

Q. What does it describe?

4

A. It describes an inflammation
of heart muscle with a virus.

6

Q. Well, just explain that to me.

7

A. Well, virus of the sort that
everybody knows, Coxsackie viruses and a number of
others can cause ---

9

Q. One of those wiggly things
you see on a microscope slide?

A. Not too wiggly; you can't
see them very well except with an electron microscope.
But this is a virus, a small particle, a small
infective particle that could start off as an
apparently benign infection like a cold or a flu
type illness and affects the heart muscle and
damages the heart muscle to the effect that it
causes death of parts of the heart muscle and death
of parts of the conduction system.

Q. Just if I can stop you there.
Is that just as a muscle may get a viral infection or
an illness, an arm muscle or a leg muscle, are you
saying that the heart muscle can get that illness as
well?

23

A. Yes.

24

25



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Q. And that is in the case of a
perfectly normal heart?

4

A. Yes.

5

6

Q. Yes. Now what happens if the
heart gets an illness in that fashion?

7

8

9

10

A. Well, if it is a very wide-
spread infection of heart muscle, that is an
extremely dangerous condition because the heart is
likely to either fail or for the conduction system
to go completely out of whack very abruptly.

11

12

Q. So what you say is that if it
is serious it may lead the heart to stop contracting?

13

A. Or to fibrillate.

14

Q. Let's get it stage by stage.

15

First of all, will it stop the heart from contracting?

16

A. It would be more likely to
fibrillate.

17

18

19

20

Q. All right. Are you aware
without going back over them whether there were
any cases in the epidemic period which exhibited
the indicia of conduction failure Type No. 4?

21

A. That is with infection?

22

Q. Yes.

23

24

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A. No, I can't recall. I

don't think there were.

4

Q. All right.

5

6

Let me ask you this about -- well,

perhaps I will just go over my list first.

7

8

If death occurs as a result of an infection in the heart, conduction failure type No. 4, will it be accompanied by bradycardia?

9

A. It may be, yes.

10

Q. Vomiting?

11

A. Yes.

12

Q. Sudden deterioration or onset?

13

A. Yes.

14

Q. Ventricular fibrillation,

15

I think you have told us about.

16

A. Yes.

17

Q. Arrhythmia?

18

A. Yes.

19

Q. Shallow respiration?

20

A. Yes. It can have seriousness because, very frequently, if you have that sort of an infection, it also affects the brain.

22

Q. Now, so far, Doctor, in addition to our first case of heart failure, we have dealt

23

24

25



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3406

Rowe
ex. (Scott)

1

K2 2 with some ten other cases of heart stoppage, nine of
3 which may operate in the case of a perfectly normal
4 heart. Have I got that right? You will take the
5 number 10 from me?

6 A. Yes, can occur.

7 Q. All these, except heart
8 failure, may occur in the case of a perfectly normal
heart?

9 A. Yes, I'm losing track of
10 the numbers.

11 Q. Well, we're down to ten with
12 conduction failure. Can conduction failures be
13 identified by post mortem?

14 A. It is extremely difficult
15 to identify them; most cases not. In order to identify
16 an abnormality of the conduction system, say, of the
17 congenital type - because that is the one where most
18 of the work has been done - an incredibly large number
19 of sections of the heart system, the heart conduction
20 system, has to be made. It is so big a job that most
21 pathologists wouldn't even begin to take it on, and
22 there are only a very few people who have had the
courage to undertake this, because it must takes months
23 to evaluate.

24 Q. Well now, let me come to
25



1

K3 2 Item No. 11, acidosis.

3 Is acidosis, independently of heart
4 defect, a cause of heart stoppage and cardiac arrest?

5 A. Yes.

6 Q. Yes. Is it well-known in
the clinic?

7 A. Yes, it's well known, parti-
8 cularly in the neonatal field.

9 Q. Yes. And would you describe
10 to the Commissioner what is meant by "acidosis".

11 A. Acidosis is really a situation
12 where the body fluids are too acid. The body fluids
13 are normally in a neutral state in terms of acid or
14 alkaline, and one can, through disturbances of various
sorts, become either alkaline or asidotic.

15 Q. Is there a balance that is
16 required?

17 A. Yes, there is.

18 Q. And is that balance as between
19 acidine and alkaline required for every cell in the
body?

20 A. Yes, it is.

21 Q. And when the balance is
22 disturbed in favour of acid, is that called asidosis
23 when it appears in that cell?

24

25



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K4

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A. Yes.

3

4

Q. And can it appear in groups
of cells and in organs?

5

A. And in the blood.

6

Q. And in the blood?

7

A. Yes.

8

Q. Now, what happens when there
is acidosis of the blood or the liver or the kidney -
let's not talk about the heart just yet? May that
have any impact on heart stoppage in a baby?

9

A. When you have acidosis, it
implies that all the organs in the body will be in
the same situation. It is unlikely that one organ
will be less acid than another.

10

Q. I see.

11

A. That's a sort of broad
generalization. So that it has profound effects on
every cell and, of course, it depends on the key
organs of the body. If you have it on your toe, it
may not be as bad as having it in the brain, you know,
that sort of impact, but it depends on how vital the
organ is. If the organ is vital, the effect is the
same on cells; that is, it interferes with the functions
of the transport of substances in and out of the cell,
on the membrane on the internal working of the energy

12

13



K5

1

2 arrangement inside the cell and just the sort of things
3 we've talked about with the boxing.⁷⁾

4

5 Q. And is acidosis a candidate
6 to induce the stoppage of heart contractions?

7

A. It can, indeed.

8

9 Q. How does it work? How does
10 that happen?

11

12 A. Well, again, it affects the
13 cellular action - that is the important thing. If
14 the cell becomes so acid that it cannot perform its
15 normal function, then the cell stops functioning.

16

17 Q. And are there cases that
18 point to that in the epidemic period?

19

A. Yes.

20

21 Q. Can that happen and lead to
22 heart stoppage in the case of a perfectly normal
23 heart?

24

25 A. It can. It would have to
be very extreme, but it can.

26

27 Q. Is there, again, the build-up
28 if you have acidosis plus a heart defect that the
29 risk to the patient is greater than with the heart
30 defect alone?

31

A. Yes.

32

33 Q. Now, when a patient has

34

35



K6

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acidosis, you have told us it can lead to the stoppage
of contractions. Is that accompanied by bradycardia?

4

A. Yes.

5

Q. I'm getting this out of
order. First of all, can this theoretically occur
where there is no heart defect?

7

A. It can, yes.

8

Q. But are you telling me it
is more likely to occur where there is a heart defect?

10

A. No. It can occur in small

11

babies who are ill or for any different number of
reasons, but it is particularly likely to occur in
babies who have lung problems or who have some meta-
bolic upset and the patients with heart disease, where
there is heart failure, there is usually a problem with
the distribution of blood around the body and, there-
fore, acidosis is a fairly common thing. In some
patients with congenital heart disease, it is even
worse than that. If you have a ductal-dependent
baby - that is, one where the presence of the open
ductus arteriosus is critical for survival - if the
conductus shuts off, one of the first things that
happens is that you get metabolic acidosis because the
blood is not being profused around the heart, and the
moment that starts, it is an inexorable course and the

24

25



1

K7 2 patient will become acidotic metabolically and that
3 just shuts down everything.

4 Q. And that leads to death?

5 A. Yes.

6 Q. And that leads to death,
7 again by the stoppage of contractions?

8 A. Yes.

9 Q. And cardiac arrest?

10 A. Yes.

11 Q. And apart from the ductus
12 that you described, there may be nothing else wrong
with the heart?

13 A. Oh, no, there usually is.

14 I'm talking about the ductal-dependent heart lesions.

15 Q. I see.

16 Now, when that occurs, is it likely
to be accompanied by bradycardia?

17 A. Yes.

18 Q. Is it likely to be accompanied
19 by vomiting?

20 A. It may be.

21 Q. Is it likely to onset
suddenly or the patient to deteriorate suddenly?

22 A. Yes.

23 Q. Is it likely to be accompanied

24

25



1

K8 2 by ventricular fibrillation?

3

4

A. If there is congenital heart disease, yes.

5

6

Q. Yes. Is it likely to be accompanied by arrhythmia?

7

8

A. Yes.

Q. Is it likely to show shallow respiration?

9

A. Yes.

10

11

Q. Is it likely to produce seizure?

12

A. Yes.

13

14

MR. SCOTT: Would this be a convenient time, Mr. Commissioner?

15

THE COMMISSIONER: Yes, until 2:30.

16

--- luncheon recess.

17

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2 --- on resuming.

3 THE COMMISSIONER: Yes, Mr. Scott.

4

MR. SCOTT: Just one matter.

5

When Mr. Lamek was examining Dr. Rowe on July 28th, Volume 18, beginning about page 3077, he drew to his attention a note --

7

MR. LAMEK: I'm sorry, what page?

8

MR. SCOTT: Well, it was dealt with in various places but I think it is dealt with at page 3077.

11

MR. LAMEK: Thank you.

12

MR. SCOTT: He drew to his attention a note, I think in the Woodcock record, which read: "could possibly be some sort of drug overdose, accidental or otherwise".

15

You will remember that that was the case of the Baby Laura Woodcock, who was I think jaundiced and a doctor whose signature couldn't be identified had made that note in the record or in the chart, and Mr. Lamek drew from that that there was, in June at least, the author of that note who was prepared to contemplate the possibility of intentional overdose of some drug and as a possible explanation, presumably, of the death of the child.

23

Dr. Rowe didn't read the note that

24

25



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2 way but Mr. Lamek apparently in the ensuing questions
3 did.

4 Now, I should tell the Commissioner
5 that we have made an effort to find out who made that
6 note, notwithstanding that his signature gave us
7 almost no clues, and we have ascertained that it is
8 a Dr. Webber, who has now returned from his vacation.

9 So that a false impression about
10 the impact of that note won't remain with you, Mr.
11 Commissioner, or with the public or press, I would
12 ask Mr. Lamek to - and we'll be glad to assist -
13 interview that doctor as soon as possible and to call
14 him out of order if necessary because the imputation
15 that his question made out is a serious one, and I
16 think it can be put to rest if Dr. Webber is inter-
viewed.

17 MR. LAMEK: Mr. Commissioner, Mr.
18 Scott has already told me about Dr. Webber's avail-
19 ability for interview and, of course, I will inter-
20 view him. So far as the imputation is concerned, I
21 refer my friend to the passage on page 3078 where
22 Dr. Rowe said to me:

23 "I don't believe he's referring to
24 the mode of death, he's referring to
25 the liver."



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And my question was:

3

"Q. Whatever he's referring to,

4

Doctor, he contemplates as a possible
explanation, does he not, intentional
drug overdose?"

5

6

In my respectful submission, the

7

8

imputation my friend suggests was plainly not left,
but it should be cleared up anyway, and I will inter-
view Dr. Webber.

9

10

MR. SCOTT: That makes the point
nicely, yes.

11

12

13

14

15

THE COMMISSIONER: All right. And
you will make him available and then this next
problem we don't have to face until after that inter-
view.

16

MR. LAMEK: That's correct.

17

18

19

MR. SCOTT: Well, one has diffi-
culties because there has been all this talk about
a mystery doctor and I just want to make sure there
is no mystery.

20

21

THE COMMISSIONER: He is no longer
mysterious! All right. That's fine.

22

23

24

25

MR. SCOTT: Q. Well now, Dr.
Rowe - if I may continue, Mr. Commissioner - we had
dealt with acidosis before lunch. I want to take you



1

2 to a word that you have used and which might form
3 the heading for my next series of questions - Apnea,
4 A-p-n-e-a.

5 Can you tell the Commissioner what
6 apnea is.

7 A. Apnea is the absence of
breathing.

8 Q. Yes. And what is the
9 origin of the absence of breathing in apnea?

10 A. It is probably a central
11 origin; that is, that there is a signal from the
12 brain that stops the respiratory drive on the
13 centre that is responsible for the rhythmic intervals
14 between breaths.

15 Q. Would that be characterized
as a neurological defect or abnormality?

16 A. Yes.

17 Q. And do I understand from
18 your answer then that the neurological defect or
19 abnormality signals the breathing to stop?

20 A. Yes. It may be a primary
or secondary brain --

21 MR. PERCIVAL: Mr. Commissioner,
22 may I compliment the technician who obviously did
23 wonderful miracles over the lunch hour because we can
24

25



1

2 all hear very well now.

3 THE COMMISSIONER: Well, that's
4 good. I'm glad that we're pleased with somebody or
5 something.

6 MR. SCOTT: If we can get Mr.
7 Percival listening!

8 MR. PERCIVAL: Mr. Commissioner, I
9 just woke up!

10 THE COMMISSIONER: Well, I will
11 pass on your compliments.

12 MR. SCOTT: That's why Dr. Rowe
13 and I will be boozing at each other for the rest of
14 the afternoon!

15 THE COMMISSIONER: I don't discover
16 things as fast as Mr. Percival and I probably would
17 have done just that.

18 MR. SOPINKA: I thought it was just
19 that Mr. Scott had lunch and he had more energy!

20 MR. SCOTT: Well now, back to the
21 case.

22 Q. Is apnea capable of existing
23 so as to stop breathing altogether apart from the
24 condition of the heart or its muscle?

25 A. Yes.

Q. And is it, in the clinic and



1

2 the literature, a well-known phenomenon?

3 A. Yes, it is, especially in
4 the context of sick infants.

5 Q. Yes. Does this have any
6 relation to the blue baby syndrome?

7 A. Not necessarily, no.

8 Q. I see.

9 Are there any cases of the 36
10 through which Mr. Lamek took you that suggest the
presence of Apnea as a cause or potential cause?

11 A. I seem to recall that there
12 are.

13 Q. Yes. Well, would you make
14 a note to put this on your list.

15 THE COMMISSIONER: But apnea, is
16 it a cause in itself? It is not a symptom of something
else?

17 THE WITNESS: It may be a symptom
18 secondarily of something else but its origin is almost
19 always attributable to the brain.

20 MR. SCOTT: Q. Well, what I am
21 getting at though is that a baby's heart may stop
22 contracting if it exhibits the neurological defect or
abnormality known as Apnea?

23 A. Yes.

24

25



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Q. Which neurological defect
or abnormality can lead to a stoppage of breath?

A. Yes.



BB
DM/wb

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Q. And it has nothing to do with
the formation or any defects in the heart?

2

A. It may have nothing to do with
it at all.

3

Q. This defect may lead to a
perfectly normal heart arresting?

4

A. Yes.

5

Q. And when that occurs is it
accompanied by bradycardia?

6

A. Yes.

7

Q. Is it accompanied by vomiting?

8

A. I don't think so, particularly.

9

Q. May its onset be sudden, or
the deterioration of the patient be sudden?

10

A. Yes.

11

Q. May it be accompanied by
ventricular fibrillation?

12

A. That would be unusual, I would
think.

13

Q. Arrhythmia?

14

A. Arrhythmia, yes.

15

Q. Shallow respirations?

16

A. Well, there is no respiration.

17

Q. No respiration at all?

18

A. No.

19

20

21

22

23

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BB2

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Q. And seizure?

2

A. Yes.

3

Q. One matter, while I have you here, one of Mr. Lamek's list is, of course, vomiting. I wonder if you could tell us something about how vomiting occurs. Most of us, I think most lay persons associate vomiting with having consumed something that upsets your stomach, or perhaps having the flu or some disorder of that type. Is that how vomiting in babies occurs exclusively, or is it -- or are its origins different?

4

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A. There are many origins to it but it can occur because of a local upset in the stomach. It can occur just if you happen to have an infection. It can occur if you have something wrong with the brain.

17

18

19

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Q. Can it occur because you have a heart stoppage, just as a seizure might occur because of a heart stoppage?

A. You may have a terminal episode where stomach contents are excreted anyway, vomiting, I suppose is a reasonable word for it, but it may not be a projectile thing, it may just be regurgitating.

Q. Item 13, Dr. Rowe, anemia, you have used that word from time to time. Will you



BB3

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describe to the Commission what you mean by anemia.

3

4

A. Well, anemia is a shortage of hemoglobin in the blood, or the capacity of the blood

5 to carry oxygen.

6

Q. Is it a disease?

7

8

A. Yes. It is a disease, because normally you have a certain amount and anemia is the reduction below that.

9

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Q. And does anemia in small babies cause a stoppage, or may it cause a stoppage of the heart or cardiac arrest?

A. It may if it is very severe.

Q. Would you tell the Commission the mechanical process by which a disease of the blood, the oxygen carrying capacity of the blood will lead to a stoppage, or may lead to a stoppage?

A. Well, that is because oxygen is essential for the activity of most metabolic things going on in cells. The actual work of the cells depends upon oxygen. If there is an oxygen shortage and especially very severe, as can occur with some types of anemia in very small and particularly newborn babies, then there can be interference with the -- solely in the function, and that will affect, as before, any organ, but the organs, the vital organs like the

heart



BB4

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and the brain are most likely to be affected.

3

4

Q. And may anemia, as a cause or contributing to a cause, so act, even if there is a normal heart?

6

7

8

A. Yes, if you have severe anemia then the heart can be damaged by the lack of oxygen, and so, the actual disintegration of cells.

9

10

Q. Can it lead to a stoppage of the heart, or cardiac arrest?

11

12

A. Yes it can, if severe.

Q. If it did, would that be

accompanied by bradycardia?

13

A. Yes.

14

15

Q. Would it be accompanied by vomiting?

16

A. I don't know.

17

THE COMMISSIONER: I am sorry.

18

THE WITNESS: I don't know.

19

Q. Would its onset be sudden, or the deterioration sudden?

20

A. It could be.

21

Q. Would there be ventricular fibrillation?

22

A. Unlikely.

23

Q. Would there be arrhythmia?

24

25



BB5

1 A. Yes, there would be arrhythmia.

2
3 There might be ventricular fibrillation because there
4 can be actual death of muscle cells in the heart and
5 that might be a source of ectopic activity of that
6 sort, but generally speaking, ventricular fibrillation
7 is unusual in small babies.

8 Q. Would there be shallow
9 respirations?

10 A. Yes.

11 Q. Would there be seizure?

12 A. There may be.

13 Q. Now, No. 14 in DiGeorge
14 Syndrome, that was an expression used, or a name that
you use in examination in chief.

15 I wonder if you could tell the
16 Commission what the DiGeorge Syndrome is.

17 A. The DiGeorge Syndrome is the
collection of abnormalities that is occasionally
18 found in young babies in which there is an absence of
19 the thymus gland.

20 THE COMMISSIONER: I'm sorry, absence
21 of the sinus did you say?

22 THE WITNESS: Thymus, T-H-Y-M-U-S
gland, and a shortage of the parathyroid gland, that
23 is absence or hyperplasia of the thyroid gland. This
24

25



BB6

1
2 gland is concerned with calcium metabolism, and the
3 thymus, of course, is important in the immune system
4 of the body.

5 In addition, the babies very frequently
6 have severe conofrontal abnormalities of the heart,
7 which means they have severe defects like truncus-
8 arteriosis, interrupted aortic arch, tetralogy if
9 you like. It is a collection of conditions in which
10 there is a threat to the patient from the heart
11 disease itself. There is a threat because of the
12 influence on calcium metabolism because they often
13 have convulsions in the new-born period because of
14 lowering of the blood calcium. In addition, they are
15 regarded as being particularly susceptible to
16 infection because of the immune system being
17 incompletely present. There is a tendency for those
18 babies to die suddenly and unexpectedly, but they
19 have enough problems there to account for a high
20 proportion of deaths.

21 Q. And what is the mechanical
22 route by which the heart stops when the DiGeorge
23 Syndrome is present?

24 A. There may be a relationship to
25 the calcium level in the blood because that disturbs
the electrolyte situation so that then the calcium



1

2

gets low and the patient develops heart block.

3

Q. Is it possible that the
DiGeorge Syndrome is perhaps more parallel to a
conduction defect?

6

7

8

9

A. I don't know that we can say
that because they very frequently have a serious
heart disease. I think, in most cases, the effect of
the non-conduction would be additive.

10

11

Q. Theoretically, is the DiGeorge
Syndrome capable of existing and causing death when
there is a normal heart?

12

A. Yes.

13

14

Q. Is it accompanied, is death
from this syndrome accompanied by bradycardia?

15

A. Yes.

16

Q. Vomitting?

17

A. It might be in some situations,
especially if they have low calcium.

18

19

Q. Sudden deterioration in the
patient, or sudden onset?

20

21

A. Yes, it has been recognized in
the literature that they may die quite unexpectedly.

22

Q. Ventrical fibrillation?

23

A. I doubt it.

24

Q. Arrythmia?

25



1

2

A. Yes.

3

Q. Shallow respirations?

4

A. Yes.

5

Q. Seizure?

6

A. Yes.

7

Q. And are there cases among the

8

36 that you and Mr. Lamek reviewed, which point to
the DiGeorge Syndrome as a cause?

9

A. Yes, there are two cases of
partial DiGeorge Syndrome, I believe, and that is
variable with the degree of severity.

12

Q. I think you have listed for me
13 cases of death, causes of heart stoppage. The
first was heart seizure, which connotes a defective
heart. Do I have it right that the other 13 can
cause heart stoppage, cardiac arrest, even in the
case of a normal heart?

17

A. Yes.

18

Q. Do I have it also that when a
number of these -- when one of these 13 diseases or
disorders exists in connection with a congenital
heart abnormality, that the risk of death is greater?

22

A. Yes, I believe that is the
case.

23

Q. And that the risk of death may

24

25



1

2

escalate as other of these causes or diseases are
3 found present?

4

A. Yes, unless they are treated
5 very rapidly and promptly.

6

Q. Now, I want to just run through
7 them again to see the extent to which their existence
8 can be determined after death by postmortem. So
9 perhaps you can just follow with me and I will read
10 them out and you will tell me if postmortem is
11 likely to show the existence of the disorder.
12 Because I always like to begin with the easiest,
13 heart failure, would the post mortem show the defective
heart?

14

A. It usually will.

15

Q. How about hypoxia?

16

A. You mean signs of hypoxia on
postmortem or just on the heart?

17

Q. When you have a death, is
18 there anything that you can look at at postmortem
19 that will help you in determining whether hypoxia
20 played a role in that death?

21

A. Yes, there are certain
22 findings at postmortem that are said to be
23 characteristic of hypoxia.

24

Q. How about sepsis?

25



1

2

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A. You would expect to be able
to find sepsis unless it was extremely early.

4

THE COMMISSIONER: I'm sorry.

5

6

THE WITNESS: Unless the death
occurred very, very rapidly after the onset.

7

Q. What would you expect to find?

8

A. You might not find anything
there then, but ordinarily you would find evidence of
infection somewhere in the body, inflammatory cells,
destruction of heart tissue and so on.

9

10
11 Q. How about respiratory illness,
12 pneumonia, atelectasis, congestion, the airway
13 obstruction, would you likely at postmortem find
14 anything that tends at points to those?

15

16 A. You would find evidence of
17 pneumonia, fluid, congestion in the lung, pulmonary
18 edema and those things are fairly standard. It would
19 depend how complete the autopsy was as to whether you
20 would find evidence as to whether there was obstruction
21 and you might have difficulty if they forgot to look
22 at the back or the oropharynx to see where the edema
23 are and that sort of thing.

24

25

Q. Yes. No. 5 was instability of
temperature or hypo- or hyperthermia. Would you find
evidence of that at postmortem?



1

2

A. I would think not.

3

Q. The next was low birth weight.

4

I take it, if you weighed the baby that would be what
you would find?

6

A. You would find if the death
occurred shortly after birth, you would have evidence

7

of a low weight, that would give that clue.

9

Q. Would there be any evidence
in the postmortem itself, apart from the weight of
the child and its age?

11

A. I'm not aware of any, there
might be others but I am not aware of them.

13

Q. And I think you have already
told us that the four kinds of conduction failure I
discussed with you would not be revealed on a
postmortem.

16

A. Very unlikely to be.

17

Q. Without the kind of testing
you have described this morning?

19

A. And even then you wouldn't
find it in some of the forms.

21

Q. No. 11 was acidosis, is a
postmortem going to reveal whether acidosis played a
part in the death?

23

A. I don't believe so.

24

25



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Q. What about apnea?

3

A. No. That's not quite true.

4

Apnea, you would not be able to identify that apnea had occurred in an individual case, unless it was something like Sudden Infant Death Syndrome that has a set of pathological findings, which, I believe, are considered by many people to be characteristic.

5

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Q. Yes, but I take it for apnea

2

plain and simple apart from SIDS ---

3

A. Yes.

4

5

Q. --- would the post mortem
reveal anything that would point to apnea as a cause?

6

A. No. You might be able to see
some toxic signs.

7

Q. Well, what about anemia?

8

9

10

11

12

13

A. Anemia, you might be able to -
you might be able to detect that from the appearance
histologically of a heart but I am not sure what
degree of certainly one would have. I think you
would have to get a pathologist to answer that one.

14

Q. All right. Now what about
the DiGeorge Syndrome?

15

A. Yes. That could be identified.

16

17

18

19

20

Q. So I haven't toted them up,
but I take it a number of these causes which may cause
or contribute to this stopping of contractions in the
heart are not causes that can be ascertained post
mortem?

21

A. Yes, some of them, yes.

22

23

24

25

Q. Now, when you are looking for
the pre-mortem you have a number of aids in that
detection process, don't you?



1

2 A. Yes.

3

Q. What are those aids just in
4 brief?

5

A. Well, the low birth weight
6 baby is obviously. Conduction failure would be the
electrocardiogram principally.

7

Q. Yes.

8

A. And the instability of
9 temperature would be the observation that the nurses
10 would provide on a regular basis.

11

Q. Yes.

12

A. And acidosis and sepsis could
13 be confirmed by measurements of blood Ph, and
culturing blood and other fluids of the body to see
14 if there is any infection growing. And anemia can
15 be determined by blood count.

16

Q. And a catheter can determine
17 the ---

18

A. And the hypoxia would be
determined by the measurement of oxygen in the blood.

20

Q. Yes.

21

A. Oxygen measurement, and heart
failure can be assessed by suitable assessment.

22

Q. Apart from those aids to
23 detection in the clinic does the observation of the
24

25



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cardiologist, the internist, the fellow, play a part in assessing the extent to which these conditions may exist?

5

A. Yes, I think it does.

6

7

Q. Can you explain how that is done? It is a matter that is foreign to lawyers, perhaps, but how do you do it?

8

9

A. Well, you do it part by obtaining historical information about the patient.

10

Q. Yes.

11

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A. That is you learn something from the family as to what has been going on in the immediately preceding time, or if the patient is transferred from another hospital, from that hospital's observation.

Then you make a physical examination which tells you rather substantially more than just that there is severe heart disease because it will allow you to assess the severity, and there are a number of ways that you can assess the severity of the heart failure in addition to the severity to the underlying heart disease.

Q. We will be coming to them later but ---

A. Yes.



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Q. --- is the rest a matter of judgment on the part of the clinician?

A. Judgment and the correct utilization of the other investigative means at his disposal.

Q. Well now there is just one point I want to make with respect to those 14 modes by which contractions of the heart may stop.

Have you any observation in the case of young babies as to whether their onset may be sudden and unpredictable? Let me show you the background and you will perhaps remember.

We have had a lot of evidence that the nurse's note indicates that Baby X is stable or nothing much has been happening for a day and then there is a cardiac arrest suddenly out of the blue in a stable course.

With respect to these 14 causes does that strike you as odd or unusual?

A. I think that babies can appear to be stable when they are not really stable and they can certainly deteriorate, some of them, there is absolutely no question about that, but it depends... Not always, but it depends very frequently on observation that is rather specialized in order



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to determine whether those babies are as stable as
you might think prior to the deterioration.

4

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That is because not everything that
is under external observation by, say, a nurse or
parent, would necessarily be sufficient guide to
indicate a deteriorating, an infant who is deteriorat-
ing who could appear stable on the outside.

9

Q. Yes.

10

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A. And that is a difficult problem
because it obviously - it would demand for the thing
to be satisfied in terms of detection of that decay
would be somebody who would be knowledgeable like the
physician, pediatrician or cardiologist in this
particular area, being able to make observations that
are different from those that I have described by
others.

Q. Now, Doctor, I want to turn

to - we dealt now with 14 causes of heart stoppage,
and I want to now turn to a list I have made apart
from those 14, of other complicating factors, and I
will tell you and the Commissioner what I would like
to deal with and then I would like to ask you some
questions under each of these heads.

The first is I would like to deal
with one's capacity to measure the severity of cardiac



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malfunction.

3

Then I would like to deal with extra-
cardiac malformations.

5

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Then I would like to deal with the
size and age of babies at time of birth as a
complicating factor. The last, with the failure to
grow and thrive in weight and height as a complicating
factor.

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Now first of all, are you familiar
with a study called The Report of The New England
Regional Infant Cardiac Program which I think was
made in February of 1980?

A. Yes, I am.

Q. Yes. And we have copies of
that. Perhaps I can show you the original and work
from a copy.

THE COMMISSIONER: I take it you are
familiar with it?

THE WITNESS: Yes, I am.

THE COMMISSIONER: Do you want the
whole thing in, Mr. Scott? You want the whole thing
in?

MR. SCOTT: Yes. This is really
only part of it, is it not, Dr. Rowe?

THE WITNESS: What I have is the



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whole thing. ,

3

MR. SCOTT: Q. Yes. I am sorry.

4

It is published in a periodical known as "Pediatrics".

5

Is that correct?

6

A. I hope the copyright has all
been satisfied.

7

Q. Well, you leave that to us.

8

We copied it; not you. Just a couple of questions.

9

THE COMMISSIONER: Exhibit 126.

10

MR. SCOTT: Thank you.

11

12

---EXHIBIT NO. 126: Publication entitled "Pediatrics",
dated February, 1980.

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MR. SCOTT: Q. As I understand it,
and I have read this document with a very low level
of comprehension which will be apparent, but I take
it its program, just to lead you a bit for the moment,
is a program that is run by all the hospitals in
I think six New England States?

19

A. Yes, it is.

20

Q. Yes. And that between 1968

21

22

and 1974 they reviewed with appropriate follow-up,
which is why they only went to 1974, some 2,251 babies
with cardiac problems.

23

A. Yes.

24

Q. Now can you describe - I'm

25



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going to take you to some of the particular parts of
it, but can you just describe to the Commissioner
what you regard as the key findings of this study?

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A. Well, this was a move in New England to try and regionalize the care of babies who were critically ill with heart disease in the first year of life; therefore it concerns only babies who come under that definition.

Q. The definition is New England babies?

A. Yes.

Q. Admitted to one of the treating hospitals?

A. Yes.

Q. And in the first year of life?

A. In the first year of life but with certain qualifications about the severity of their heart disease.

Q. Fine.

A. These were - I believe that they - I think they had to undergo cardiac catheterization and endocardiography, be operated upon or have died to be included in the total.

In other words they would not admit a baby who was quite well with a minor defect. So



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these were - they call them critically severe meaning
that they were severe enough that they either died,
had to have an operation or had undergone catheter-
ization or death.

3

4

5

Q. These were the babies that
they reviewed?

6

7

A. Yes.

8

9

Q. And does the study reveal
anything about the survival rates or capacities of
those babies?

10

11

A. Yes, it does.

12

13

Q. Can you tell the Commissioner
what it discloses?

14

15

16

A. Yes. It summarizes the fact
that the mortality is between 35 and 40% in all these
infants.

17

18

19

20

Q. Now just let me see if I
understand that. Does that mean that of the babies
in this category who were sufficiently ill to undergo
catheterization or surgery, 35 to 40% died or 35 to
40% died in the first year?

21

22

A. Died in the first year I
believe.

23

24

25

Q. All right. So the study
doesn't tell us to what extent babies in this



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category die in succeeding years?

3

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A. Not directly I think. There may be some tables on that but I think their conclusion is concerning the first year.

6

7

Q. Now did it tell us anything about the various determinants for survival?

8

9

10

A. Yes. This was one of the important parts of the examination. They looked at factors that might be important in the survival and came to conclusions as a result of that analysis.

11

12

Q. Can you summarize the conclusions for us?

13

14

A. Well, as would be no surprise to anybody, the extent of the anatomic abnormality was of considerable importance.

15

16

17

Q. Well, if I can stop you there, did they make an effort to characterize the severity of the anatomic disorder?

18

19

A. Yes. They did do some grading and did analysis of the severity in that way I believe.

20

21

22

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Q. And did they provide any figures as to the extent to which a severe anatomic disorder led to death apart from the 35 to 40% in the first year?

A. Yes, they did that for certain



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malformations. For example, transposition of the
great arteries.

3

Q. And what did they find out?

4

Is there a chart that you can point us to?

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A. I think there is a chart on
Table 27 on page 401.

Q. Yes.

A. That just gives the different malformations of the heart and the first month's mortality and the first year's mortality.

It is not quite what you are asking for but that is the first table I can see.

Q. Well, let's see if I can read that.

What it shows is -- it lists the various kinds of heart deformations down the left-hand side of the page.

A. Yes.

Q. And then it lists the number of infants in the study who were shown as disclosing that kind of disorder.

A. Yes.

Q. And then the third column, do I read that as the number from that category who died in the first thirty days?

A. Yes. This is in relation to surgery.

Q. Yes.

A. So, it is not quite as --



1
2 DD2 They have tables on the left-hand column for
3 mortalities for medical management and mortalities
4 for surgical management on Table 20/395.

5 Q. Well, just so we can see
6 how we follow it up and then we'll go to some of the
7 other changes, this Table 27, for example, shows that
8 if the baby had had a ventricular septal defect,
9 there were 113 in that study, 20 of them would have
10 died if they were to be surgically managed within
the first 30 days.

11 THE COMMISSIONER: No, I think that
12 is percentage.

13 MR. SCOTT: I'm sorry.

14 THE WITNESS: 20 per cent.

15 MR. SCOTT: Q. 20 per cent would
16 have died and 23 per cent would have died in the first
year.

17 A. Yes.

18 Q. Is that an additional 23 or
19 does that include the original 20?

20 A. No. That's 23 per cent.

21 Q. And therefore if you take
22 all the defects that were surgically managed you
23 come out to a first year mortality rate of 40 per
cent averaged at the bottom?

24

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DD3

2 A. Yes.

3 Q. Now, is there a table that
4 deals with infants who are not surgically managed?

5 A. I'm sure there is.

6 Q. Well, take your time.

7 A. I think Table 20 is probably
it. It is probably the easiest one to look at.

8 Q. All right. And there I take
9 it --

10 MR. LAMEK: I'm sorry, what number?

11 MR. SCOTT: Table 20 at page 395.

12 Q. Again, the deformations are
13 listed under two headings, "Medical Management" and
14 "Surgical Management", and the averages for crude
15 mortality in each case is 40 per cent, which means
16 40 per cent die within the first year. Have I got
that right?

17 A. Yes. I can't remember what
18 the crude versus the adjusted, whether they excluded
19 certain things in the adjustment.

20 Q. All right.

21 Well now, apart from showing those
22 gross mortality figures which you described earlier
23 in summarizing the reports as saying 35 to 40 per cent
24 of babies with these deformations died in the first

25



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2 year, I then asked you if there were determinants
3 that the report dealt with with respect to survival,
4 and the first thing you dealt with was the severity
5 of the anatomic diagnosis.

6 A. Yes.

7 Q. Now, does the report set up
8 a system for measuring severity of anatomic diagnosis?

9 A. Well, the system they used
10 is an arbitrary one and their conclusion that when
11 they looked at the validity of their prognostic
12 categories, as they call them, they were found to be
13 reasonable but I don't know what the evidence is, the
14 statistical analysis.

15 Q. Did the study tell you anything
16 about the relation of mortality to the age of
17 admission?

18 A. Oh, yes, that was a very
19 important observation.

20 Q. And what did it tell you?

21 A. Well, that showed that the
22 majority of deaths occurred in babies who were
23 admitted during the first two months of life.

24 Q. And do you have the per-
25 centages?

26 A. I think it was somewhere around

27

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1 DD5

2 55 per cent or something like that. I can't see it
3 here.

4 Q. Well, can you take a moment
5 and put your finger on it in the report? We could
6 even take perhaps a short minute break if that is
7 necessary. You were good enough to give the figures
8 to me, I've got them but I wanted you to place them
in the report, if you can.

9 MR. LAMEK: Table 41, I think, isn't
10 it, Doctor?

11 THE WITNESS: I think that is probably
12 the one.

13 MR. LAMEK: Age of admission is
the determinant for survival.

14 MR. SCOTT: I want the percentages.

15 MR. LAMEK: It is given us the
16 percentages. 51 per cent of two months.

17 THE WITNESS: Table 41, I think
18 Mr. Lamek has given it.

19 MR. SCOTT: Q. All right. What
does that show?

20 A. It shows that in the first
21 two months the mortality is about 50 per cent; it is
22 49 per cent in one time span and 51 per cent in
23 another and then, after two months, the risk is lower,

24

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Rowe
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substantially lower.

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Q. Does that mean that of the
40 per cent who will not survive the first year, 50
per cent of that 40 per cent will die in the first two
months?

6

A. I haven't done the figures
but I would think that it means that the greater
contribution of the overall one-year figure is made
in the first two months.

10

Q. Yes.

11

Now, is there a table that tells
12 us the relationship between the age at which you
submit to surgery and the mortality?

13

A. Yes, there is. Table 42.

14

Q. And what is the figure there?

15

A. Well, the figure there is
16 that, in the first two months of life, the risk of
dying from surgery is very much higher than after that
17 age. The risk is somewhere around, again, 50 per cent
18 for the first two months and about 20 per cent in the
19 subsequent months of the first year.

20

Q. All right.

21

So, can I summarize it this way. In
22 round figures, in dealing with cases of reasonably
23 severe anatomical diagnosis, roughly 40 per cent of

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DD7

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babies will not survive their first year of life?

2

A. Yes.

3

Q. Dealing with those same
babies, roughly 50 per cent of them, whether they
are surgically managed or not, will die in the first
two months of life?

4

A. Yes.

5

Q. Now, is there a table that
tells us the contribution of birth weight to the
statistics?

6

A. Yes, there is. Table 43.

7

Q. Yes. And what does that
tell us?

8

A. Well, that tells us that
smaller babies have a higher mortality. They have
used the cutoff point of 2 kilograms.

9

Q. You see, you have been saying
for two weeks to Mr. Lamek that smaller babies are
inclined to die faster and what I am trying to do here
is to see, apart from your judgment, if there is
evidence that sustains that and perhaps you can give
me what the figures were in this study.

10

A. Well, the figures that they
arrived at were that if you were less than 2 kilograms,
the risk of dying before the first year was about

11

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DD8

2 50 per cent.

3 Q. Yes.

4 A. If you were over 2 kilograms,
5 the risk of dying was 39 per cent, or 40 per cent.

6 Q. Yes.

7 A. Not a huge difference but it
is a difference.

8 Q. Is that a risk that is --
9 Is the weight factor a weight factor in this study
10 that is applied to a reasonably severe cardiac
11 malformation case?

12 A. I'm not sure. The 2 kilo-
13 grams is a bit lighter than I would have expected, but
14 the figures show that. I would have to get someone
15 to look at the material to see whether or not there is
that big a difference.

16 Q. Do you mean to say that you
17 would have thought that there would be more babies
18 dying who weighed less than 2 kilos?

19 A. No. I would have thought the
20 cutoff point might be a bit higher than 2 kilos.

21 Q. At what, say?

22 A. Well, say, at 2.5. But I
23 think that some biased statistician might be able to
24 look at that.

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Q. So that what we can say is that, on these three factors that we have looked at as applied to reasonably severe cardiac malfunctions, there is the production of a survival rate of 60 per cent for the first year, half of those more or less dying in the first two months?

A. Yes.

Q. Now, are there figures or comments in this report that deal with another factor; that is, extracardiac malformations?

A. Yes. They have a section on that which is on page 408.

Q. 8 or 80?

A. 408.

Q. Yes.

A. And the conclusion of that was that the presence of an additional major malformation is usually a direct affect of survival.

Q. Does the report - I interrupted you - tell you anything about the extent to which an extracardial malformation is likely to be found in a baby with a cardiac malformation?

A. Yes, I believe there is a figure on that, and I don't know where that is. This group has published separately many, many papers on



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2 DD10

the association of congenital heart disease with
extracardiac malformations, and the usual figure that
is given for this is somewhere between 25 and 30 per
cent.

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Q. All right. Now, what does
that mean exactly? If you take a baby who has a
cardiac malformation, does that tell you anything
about the likelihood of another system being mal-
formed?

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A. Yes, because the figure is
somewhere around 25 or 30 per cent. That means that every
clinician who is concerned with a baby who has
heart disease is looking to see whether there is an
additional anomaly.

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11
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13

Q. And he will know that, based
on those figures that, say, in one of three cases
there will be an extra anomaly?

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16

A. Yes.

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Q. And are there any figures

in the report that deal with mortality when there is
more than one anomaly in a system? You have the
cardiac anomaly and then you have the 30 per cent,
I think it is actually 28 per cent where there is
an extra anomaly. What does that do to the death
rate?

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DD11

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A. Well, I'm not sure about the rise in numbers. You may have missed that, but there is no question that with very severe anomalies, very severe additional extracardiac anomalies, the mortality is gravely affected. In fact, in this study, they excluded those patients from the mortality conclusions by calling them an adjusted mortality after they had excluded those patients. But I can't find the spot specifically that, whether if you have five, you have more risk than if you have two.

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Q. Yes. Well, is there any sense in the medical profession on that subject?

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A. I think the more important
thing is the nature of the extra cardiac anomaly.
If you have only one major additional anomaly and
it happens to be in the brain then that's much more
important than if you have a turn in the little left
finger.

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Q. Let's take each of those
subjects, and I want to ask you first of all about
the severity of cardiac malfunction. This report
makes an effort as you have described to gauge the
severity of the malfunction.

Now, you have given evidence, Doctor,
that in the 36 babies that were examined by you and
Mr. Lamek, I think all but three babies Pacsai,
Hines and Hayworth exhibited a congenital structural
abnormality of the heart?

A. Yes.

Q. So of the 36 I think you
were looking at 33 had a cardiac malfunction of some
type?

A. Yes.

Q. Now would you be able to
gauge within reasonable medical limits the severity
of those 33 heart defects?

A. Yes.



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Q. Can you tell the Commissioner how you would approach that exercise of measuring the severity, because when we know their severity then we can take the New England figures and see how many of them would have died had they lived in New England. What is the process by which you would gauge the severity of those malfunctions, cardiac malfunctions?

A. Well you would look at the -

I think the cornerstone of that would be the anatomic abnormality.

Q. Yes.

A. The precise definition of the abnormality in the heart itself.

Q. Yes.

A. Because there are certain malformations in whom the delineation of the detail tells you immediately that this is such a severe malformation that death is inevitable.

Q. Yes.

A. So that would be an example of one extreme in the situation.

In another you might find a baby who has a ventricular septal defect which might be of moderately large size. So you would be able to say



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that the anatomy was moderately severely distorted
and you could tell, depending upon the age of the
patient how well that heart was functioning that you
could make predictions as to the size of the defect
in relation to the size of the child, what the probable
outcome for that baby might be. Not in terms of
immediately whether it is going to die or anything
but in terms of the prognosis generally.

3

Q. Well, I'm going to ask you
to do this in due course, but I just want to make
sure that you think you can do it.

4

If you take the New England statistics
that in moderately severe cases 40% of babies with
moderately severe cardiac malformation do not survive
one year?

5

A. Yes.

6

Q. If you take that figure, do you
think you can characterize the babies about which we
have been talking with sufficient certainty to apply
that figure from the New England standard to them?

7

A. I think you probably could.

8

THE COMMISSIONER: Would this be a
good time?

9

MR. SCOTT: Yes, Mr. Commissioner.

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THE COMMISSIONER: We will take

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15 minutes then.

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---Short recess.

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---Upon resuming.

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THE COMMISSIONER: Yes, Mr. Scott.

6

MR. SCOTT: Thank you, sir.

7

Q. Let me come back, Dr. Rowe,
it's just occurred to me to ask you to do something
that probably is not possible to do. You told us
that the New England report showed that of the
babies in their study 35% to 40% did not survive the
first year, have I got that right?

12

A. That is correct.

13

Q. You also told us that age
and admission had a bearing because 54% - I'm sorry,
51% of those babies died in the first two months of
their life.

16

THE COMMISSIONER: That is 51% of
the 40%?

18

MR. SCOTT: Yes.

19

THE COMMISSIONER: That is from
which table?

21

MR. SCOTT: I haven't got the table
numbers.

22

MR. SOPINKA: Table 41, page 407.

23

MR. SCOTT: Q. You also told us

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that the study revealed that extra cardiac malformations occurred in about 30% of the babies, is that right?

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A. I couldn't find their figure you recall.

7

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Q. All right. Well, we can leave that, that was your assessment.

9

10

A. My understanding was it was somewhere around there.

11

12

Q. And I think you also told us that one of the tables show that 56% of the babies died, if operated on, in the first two months?

13

14

A. Yes.

15

16

Q. And the last thing you told us was that the ---

THE COMMISSIONER: I'm sorry, that

is Table 42 I guess, 56%?

17

THE WITNESS: I think so.

18

MR. SCOTT: Q. Yes, it is, sir.

19

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A. That is 50% if you take both time periods.

21

22

23

Q. And then you also told us that the report presented I think at Table 39, a method for measuring the severity of cardiac malformation?

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A. It is an arbitrary method,

yes, I think it has some problems but it is reasonable.

Q. It fundamentally divides the

cardiac malformations into various categories and

groups them with respect to severity moving from

Group 4, which is the most severe to Group 0 which

is the least severe, is that right?

A. Yes.

THE COMMISSIONER: What is that table

please?

THE WITNESS: That is Table 39.

MR. SCOTT: It is a linear table

rather than a graph.

THE COMMISSIONER: Yes, I see.

MR. SCOTT: Q. And then you also gave us figures and I don't again have the table number, on birth weights.

MR. SOPINKA: Table 43.

MR. SCOTT: Q. Table 43 I am told that that affects mortality.

A. Yes.

Q. Now then, I asked you if you thought you could take the 36 cases which you have discussed with Mr. Lamek and measure the severity of cardiac malformation applying generally the standard



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that was used in the New England report, and I think
you indicated that you could do that?

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A. Yes.

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Q. And is it possible, in your
judgment, having done that to make a reasonable
judgment as to the severity of cardiac malfunctions
in those cases?

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A. Yes, it would be possible to
do that, in two ways. One to do it on the condition
prior to death.

9

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Q. Yes.

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A. And the information available
to us prior to the death of the patient, and then it
could be done with slightly different results from
those of whom we have information available in post
mortem.

THE COMMISSIONER: I am sorry, I am
having trouble, I am just not sure what it is he
is going to do.

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MR. SCOTT: He is not going to do
anything yet, I am just going to ask him if he can
measure the severity of those, of the 36 babies ---

THE COMMISSIONER: Yes.

MR. SCOTT: --- applying the New
England, roughly the New England standard for severity.



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2 THE COMMISSIONER: That is Table 39?

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MR. SCOTT: Q. Yes. If he can make

that judgment with a reasonable degree of confidence about 36 babies who died, and I want him to make that judgment before their death. That is to say I don't want him to look at the autopsy. I take it Dr. Rowe, that you may, it is conceivable that you might make a judgment that a baby's formation was not particularly severe only to find after autopsy that you were wrong and that an examination of the heart showed it be very severe.

A. It would be most likely in that area that there would be a difference.

Q. So what I wanted to know,

whether you could do it with any confidence, is judge how you would rank the babies with regard to severity of cardiac malformation before their death. We have spent a lot of time looking back and second guessing what we might have thought. I want you now to begin to make judgments as you would have made them in the clinic July, August through March 1981.

THE COMMISSIONER: Are you doing this for its own sake or is this leading to something?

MR. SCOTT: It is leading to something.

THE COMMISSIONER: Have you told us



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what that something is yet?

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MR. SCOTT: It is going to show that the judgment of the cardiologists as to severity of these illnesses led to a conclusion that a number of the babies might die.

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THE COMMISSIONER: Oh, I see. The idea being if all 36 of them came in diagnostic Group 4 it would be a reasonable assumption that they might all die, is that it?

10

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MR. SCOTT: Well, to be frank, one of the points I think Mr. Lamek has made, and particularly with respect to the September conference is you had 10 babies who died in July and August.

14

THE COMMISSIONER: Yes.

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MR. SCOTT: That graphically is more than you had in April and May, why didn't you do something about it? In fact I think that is the word Mr. Lamek used. The answer to that question is governed by how you looked at those deaths, if you regard them as severe cardiac cases in which death was 70% risk then you have an elevated level of deaths, but you have another high point like one of the dozens of graph but it is a great misfortune.

THE COMMISSIONER: But surely he can use the autopsy results if they were available in



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September, could he not?

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MR. SCOTT: Well, I will ask him to do that in due course, but I have to take it stage by stage.

6

THE COMMISSIONER: All right.

7

MR. SCOTT: I am very slow about it.

8

9

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THE COMMISSIONER: If you wanted to have a prediction if it is for the purpose of showing the propriety of his actions in September and December or January, because then he surely should have available to him all the information he had available at that time.

13

14

MR. SCOTT: Yes, we will come to that.

15

THE COMMISSIONER: Yes, all right, I am sorry.

16

MR. SCOTT: Thank you, sir.

17

18

19

Q. Now the second thing is extra cardiac - first of all we have established that you can make that judgment about severity, am I right?

20

A. Yes.

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Q. Now let's come to extra cardiac malformations and I will find it for you later in the New England report. My understanding is that 28% of their babies exhibited additional



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malformations beyond heart.

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I want to ask you, first of all,

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if you can ---

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MR. LAMEK: I just wanted to tell

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you that was page 392.

7

MR. SCOTT: Q. It is really Table
18, isn't it, Dr. Rowe?

8

A. Yes.

9

Q. It shows the relation between
10 extracardiac anomalies and diagnostic categories
11 running from 63% down and averaging at 28%. I take
12 it for the babies in the - among the 36, you can
13 make a judgment about the extent to which they
14 exhibited extracardiac malformations.

15

A. We can.

16

THE COMMISSIONER: This figure,
17 this Table 18 if I understand it correctly does that
18 say an average around 28% of babies with cardiac heart
19 defects of one sort or another have extracardiac
anomalies?

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3 MR. SCOTT: That is the way I
4 read it. Perhaps I had better ask Dr. Rowe.

5 THE WITNESS: That is correct.

6 Q. And I think you have also
7 told us - it's at page 394 --

8 THE COMMISSIONER: But before we
9 go into this, I don't understand it. Table 18,
10 endocardial cushion defect. I am not too sure - in
11 fact, I am sure I don't know what that stands for,
12 but they say 63% of the total. What is that total of?

13 THE WITNESS: 63% of 119.

14 THE COMMISSIONER: Oh, I see. The
15 119, that particular defect?

16 THE WITNESS: Yes.

17 THE COMMISSIONER: 63% of them had
18 extracardial defects.

19 THE WITNESS: Cardiac anomalies.

20 THE COMMISSIONER: I see.

21 THE WITNESS: The reason for that
22 very high figure there, Mr. Commissioner, is that the
23 majority of patients with endocardial cushion defect
24 have Down's Syndrome so this high association.

25 MR. SCOTT: Q. The significance
of this, Dr. Rowe, is revealed at page 394 where the



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report says, if I read it correctly, second sentence,
"effect on survival":

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"It was shown that for infants with
severe extracardiac anomalies there
was a significantly higher mortality
than for infants without associated
anomalies."

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A. Yes.

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Q. So that if you had only a
cardiac malfunction, your chances of surviving the
first year or of dying the first year was 40%. If you
had a severe extracardiac anomaly on top of that, your
chances of dying went up to 48%.

A. Yes.

THE COMMISSIONER: These are not
figures you are getting from the table? These are
figures that you are --

MR. SCOTT: I am getting from the
text of the study at page 394.

THE COMMISSIONER: Yes, I see.

MR. SCOTT: Q. And can I ask you,
doctor, is that consistent or inconsistent with your
general observation in the clinic or in the hospital
about the effect of extracardiac malfunction?

A. I think what they are doing



Rowe
ex. (Scott)

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FF3 2 there, though, is they are accepting as -- they are
3 accepting only the highest grade of extracardiac
4 malformations as having a major influence, and they
5 say without associated anomalies.

6

Unless I am mistaken that means
the milder or more moderate forms of malformation.

7

I think those things may have an
8 additional effect or importance on occasion as well,
9 but that is the data from their study and the way
10 they have set it up, so I am prepared to accept that.

11

For example, if I may amplify on
that just to get the point?

12

Q. Yes.

13

A. The baby who has tetralogy
14 of Fallot and who has a bad bilateral harelip and cleft
15 palate would not be graded in this study as being of
16 high risk.

17

Q. What would you say?

18

A. I would think this is a high
19 risk situation because the baby who has got tetralogy
of Fallot and is therefore blue is going to have a
20 great deal of difficulty with a lot of mucus in the
21 throat and constant suctioning so this is the sort of
22 baby that is very liable to have an airways obstructive
23 type of problem that could very well be critical to

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Rowe
ex. (Scott)

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2 survival.

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or two.

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Q. So the report is more
conservative than you --

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A. I would have thought so.

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Q. They take into account in
moving the figure from 39% to 48% only as you have
said severe extracardial malfunction?

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A. That is the way it appears
to me, yes.

16

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Q. What would you say about
the less than severe extracardial malfunction as playing
a role in mortality of babies with cardiac malfunction?

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A. I think obviously mild extra-
cardial anomalies, like an extra toe or something like
that, are not going to have any effect but I think
there are a number of more moderate group of mal-
formation, of extracardial malformations, that might
conceivably have quite an effect.

Q. Can you just tell the



Rowe
ex. (Scott)

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FF5 2 Commissioner what they are in your opinion.

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Commissioner what they are in your opinion.

A. Principally conditions which affect the respiratory system. The cleft palate and harelip is one. Trachialesophageal fistula; dia-phragmatic hernia is another. In other words there are a number.

Q. Can you list any others --

A. I can formulate a list for you if I sat down and thought about it.

THE COMMISSIONER: These are a list --

Are these the severe --

MR. SCOTT: No, these are --

THE COMMISSIONER: Table 19 seems to list -- I take it Table 19 are the ones, are they not?

THE WITNESS: Yes.

THE COMMISSIONER: Those are the ones they do take into consideration, do they not, Table 19?

THE WITNESS: They just give a broad sweep, but if we kept to that sort of list, Mr. Commissioner, it would mean respiratory anomalies might not be graded by the New England study as severe. But they I think -- in my view, respiratory anomalies can be important in prognosis.



FF6

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2 MR. SCOTT: Q. The point I want
3 to get at is when the New England study moves the
4 death rate from 39% to 48% because of a severe
5 extracardial malfunction, you have said that your
6 reading of the report leaves them to define severe
7 extracardial malfunction by excluding malfunctions
8 that you would have included as having an effect?

9 A. As maybe having an effect, yes.
10 There is a risk factor which you would have to assess.
11 I think they are doing a broad sweep and I wouldn't
12 quarrel with the way they have done it but I think
13 there are definite situations where the presence of
14 a less than severe extracardial malformation can have
15 a significant effect on the outcome.

16 Q. Let me turn to one other
17 factor.

18 THE COMMISSIONER: Before you go
19 any farther, I am not - and please don't misunderstand.
20 I am not arguing with you, I am trying to get it
21 through my head where you are leading us. It would
22 seem to me that it would naturally follow that a baby
23 who was sick with heart disease of some sort, if he
24 had something else wrong with him, it certainly wouldn't
25 be a help. It would be worse.

Now, I can well understand that the



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2 FF7 more serious it is, perhaps the more likely it will
3 make that baby die. But what is all of this leading
4 to? Are you going to try to transpose these per-
5 centages in some way on to the 36 babies that we
6 are investigating?

7 MR. SCOTT: What I seek to show,
8 Mr. Commissioner -- let me put it this way: What has
9 been said is that in the epidemic period there was
by graph an elevated level of deaths.

10 THE COMMISSIONER: Are you going to
11 try to show to us in the epidemic period there was
12 also extracardial defects?

13 MR. SCOTT: What I am going to
14 try to show to you is that the babies who died in the
15 epidemic period - this has nothing to do with whether
16 there was an intentional homicide; I have nothing to
17 say about that at the moment - but the babies who
died in the epidemic period were very high risk babies.

18 THE COMMISSIONER: But there may
19 have been the same kind of babies in the other --

20 MR. SCOTT: There may have been.
21 If my friend shows that or somebody else shows that.
I am simply showing --

22 THE COMMISSIONER: It doesn't tell
23 me anything unless I know what the other babies were

24
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FF8

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as well.

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3 MR. SCOTT: Well, it may be that
4 someone will tell us about the other babies.

5

THE COMMISSIONER: You see --

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7 MR. SCOTT: It may be that even
8 I will tell you about that but what I seek to show you
9 now is -- well, let me put it this way: Mr. Lamek's
10 questioning - I almost said evidence - was advanced on
11 the theory that you had a baby who was stable, who was
12 about to, in some cases, the way he put it, you thought
13 was about to be sent home and all of a sudden - his
language; not mine - all of a sudden there was an
incident and the baby died.

14

THE COMMISSIONER: Yes.

15

16 MR. SCOTT: Now that bears examina-
17 tion. I have gone at it one way by showing the
18 indicia of suddenness is not to be relied upon. I
19 think it is also helpful to show you, sir, if I can,
20 that these babies who died were to a very high degree
21 likely to die because of the conditions, and when Dr.
22 Rowe said that to Mr. Lamek, Mr. Lamek challenged
23 him; wouldn't accept that, and that of course is what
24 this evidence is led to.

25

THE COMMISSIONER: Well, all right.

23

Thank you. I am having some difficulty in putting

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Rowe
ex. (Scott)

FF9

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2 together the New England figures with our own figures.
3 Maybe I am just misunderstanding.

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MR. SCOTT: We don't have those

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figures, sir. For example, if you take the New
England figures that show 50% of the babies defined
in a certain category died within the first year --

THE COMMISSIONER: Yes.

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MR. SCOTT: -- we can only produce
the comparable figure by analyzing all the babies,
hundreds, who went through the ward in the relevant
period of time. And then we would have a figure that
was the same, more or less, than the New England
figures.

We might indeed have a figure that

showed that our mortality rate at the Toronto Sick
Children's Hospital was substantially less than 48%.

We haven't done that exercise, and

it is an exercise of very considerable scope, and I
am not sure that we are going to be able to do it. So
short of that, what I am saying to you is I think it
can be demonstrated that these babies, many of them,
not all of them, were very severely ill and likely to
die.

If everybody accepts that proposition

I have nothing more to say.



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FF10

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THE COMMISSIONER: Oh, no, but I
don't think I can make that offer to you that every-
body does accept the proposition. Certainly, though,
Dr. Rowe has given us evidence that these babies
were suffering from certain cardiac and other mal-
formations as a result of which their deaths were not
surprising.

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What I am trying to find out is how
we are particularly helped by the New England report.

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MR. SCOTT: You are helped by the

New England report because the first thing it shows
is that babies in this category -- Dr. Rowe has been
saying --

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THE COMMISSIONER: Babies with

extracardiac problems would die more likely more
quickly than babies with just cardiac problems.

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MR. SCOTT: What it shows is that

almost 50% of babies with these difficulties in
children's hospitals in New England, which includes
the Boston Children's Hospitals, one of the great
hospitals of the world, 50% of them die. Now, that
it seems to me is a fact that is demonstrated and
the variables that can be introduced, such as extra-
cardial malfunctions and so on, show how those
extra anomalies add to the figure.

Now the first thing we begin with



Rowe
ex. (Scott)

FF11

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2 is that there is no suggestion that anywhere near
3 50% of the babies in Toronto Sick Children's Hospital
4 died in this relevant period. Our percentage is
5 way below that. No doubt about that.

6 We have a ward that will hold 38
7 over the epidemic period. So our gross figures are
8 different than the New England figure.

9 Then you take the group of babies
10 who regrettably died and you look to see whether they
11 were, because of their malfunctions, candidates by
12 virtue of their conditions and the indicia that the
13 New England study reveals, candidates to die within
14 the first year.

15 Now, I hope that is helpful and if
16 it is not I don't want to...

17 THE COMMISSIONER: No. I'm sure
18 it is helpful, Mr. Scott, but I think you must under-
19 stand that all I am doing is trying to make sure that
20 your brilliant advocacy is getting through, that is all
21 to me, and so to understand --

22 MR. SCOTT: Well, as long as your
23 lordship and I understand each other we don't have to
24 worry about the others. Let them look after them-
25 selves.



Rowe, ex.
(Scott)

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THE COMMISSIONER: Well, my ignorant questions might help them to avoid some of their own. So, we'll see.

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MR. SCOTT:Q. Well, let me ask you, Dr. Rowe, if you can refer now to page 396 of the report. Under High Risk Factors is there a reference there to the size and age of babies at time of birth?

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A. Yes, there is.

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Q. And what does it say about

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that risk?

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A. It says that low birth weight was found to affect survival regardless of all other factors.

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Q. All right. Now, that's the third factor. We've had the severity of cardiac *? malformations*, renal function, we've had the presence of extra anomalies, we've had the size and age of babies at the time of birth. Now, I want to ask you about the failure to grow and thrive in weight and height and I want to ask you if that terminology refers to a phenomenon that is recognized in cardiology?

21

A. Yes.

22

Q. And what does it mean?

23

A. This means that babies don't grow properly. They don't grow, they don't put on

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weight, and they don't elongate.

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Q. And what is the impact of
that in terms of a baby with these other deficiencies,
cardiac anomalies and additional anomalies?

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A. Well, the most obvious feature
is that they're very thin and skinny and they have
very little reserves in terms of fat or any of the
usual energy reserves that babies have. So, they
are at high risk for running short of fuel, as it
were, if they are stressed in any further way; stressed
by infection or stressed by an arrhythmia, stressed
by progression of the disease and so on.

Q. All right. Now, with respect
to these four factors, did you at my request make a
review of all 36 babies?

A. Yes.

Q. Yes. And did you review those
babies with the four factors I have elicited in mine?

A. Yes.

THE COMMISSIONER: Those four factors,
so that we all know what they would be.

MR. SCOTT: The severity of cardiac
malformation.

THE COMMISSIONER: And the non-cardiac.

MR. SCOTT: The presence of the



Rowe, ex.
(Scott)

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GG3 non-cardiac anomaly.

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THE COMMISSIONER: Low birth weight.

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MR. SCOTT: Size and age and time of
birth and failure to thrive.

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THE COMMISSIONER: It would be size
and weight, not age.

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9

MR. SCOTT: Thanks.

THE COMMISSIONER: And the failure

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to grow and survive, yes.

11

12

MR. SCOTT: Yes.

THE COMMISSIONER: So, that's cardiac,
non-cardiac, birth weight and growth weight.

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MR. SCOTT: Q. Now, have you got that
in printed form?

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A. Yes, I have.

16

17

MR. SCOTT: Now, I don't have copies
of this for everybody at this stage, so, we can
undoubtedly get it.

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THE COMMISSIONER: Well, it's pretty
close to closing time anyway, so, we could - I don't
know, it will probably take some considerable time to
go through this, would it not, for each one of these
children? It might be an idea to have copies made.

23

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25

MR. SCOTT: Perhaps I can ask some
questions about it that won't need the document.



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(Scott)

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GG4

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THE COMMISSIONER: No, no, by all
means, yes.

MR. PERCIVAL: Unfortunately,

Mr. Commissioner, the questions are going to mean
nothing to the rest of us.

THE COMMISSIONER: Well, that's
what I thought.

MR. SCOTT: But I said I thought I
could ask some questions without the document.

THE COMMISSIONER: Mr. Scott has
promised to ask good general questions that are going
to mean a great deal to us.

MR. PERCIVAL: We wouldn't need the
document you mean?

THE COMMISSIONER: We won't need the
document at all. I promise, I won't even look at
mine so that I will know when ---

MR. LAMEK: Now you're in trouble,
Scott.



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2 THE COMMISSIONER: Well, if we copy it
3 we may as well rise for the day but if you can ask
4 some general questions that will be of assistance, let
5 us have them.

6

7 MR. SCOTT: Q. Well, first of all,
8 Dr. Rowe, do I understand that this study was prepared
9 in the last couple of weeks?

10

A. Yes, it was.

11

Q. And it was prepared in the
12 light of the analysis in the New England Journal, is
13 that correct?

14

A. Yes, it was, with the exception
15 that the severity of malformation was handled a little
16 differently on a number of additional factors.

17

Q. But it is with that proviso
18 an attempt to model an analysis on the New England
19 study?

20

A. Yes.

21

Q. And do I understand that at my
direction, perhaps unwisely, I have to take the
responsibility for this, I think it was prepared as if
you were looking at the patients before their death.

22

A. Yes.

23

Q. And do I understand that Dr.
Freedom was also asked to do the same thing?

24

A. In respect to the predictions,

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GG2.2

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yes.

BB/wb

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Q. Yes. And that you and he did not compare your analyses before they were completed?

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A. No.

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Q. Now, perhaps I will have to start looking at the form now, Mr. Commissioner, if you want to stop.

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THE COMMISSIONER: I take it we're not going to have Dr. Freedom's document until we have Dr. Freedom. I take it he has a different document, he's prepared a different document.

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MR. SCOTT: Well, he did the same exercise, applying the New England Study to this exercise. He did it and we asked him to do it alone, we didn't want Dr. Rowe and Dr. Freedom comparing notes as they went along. My friend can have his result anytime he wants it.

THE COMMISSIONER: That's fine. No, no, no reason to use it now. I think we could probably, if there is nothing else you want to ask until we go over to the document itself, well then, let's --

MR. LAMEK: Mr. Commissioner, can I suggest we copy it here so Counsel can take it away with them. We can take it up to the 22nd Floor and



GG2.3

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have it copied immediately.

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THE COMMISSIONER: All right. Well,

I think Counsel can just drop in on their way home.
It's a funny way to go home, but they can do it, go
up to the 22nd Floor.

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MR. LAMEK: I go home that way every

night.

9

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THE COMMISSIONER: All right. Well
then, until 10:00 o'clock tomorrow. Have you any
thoughts for us, Mr. Scott, of how long you think
you might take?

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MR. SCOTT: A full day.

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THE COMMISSIONER: But just all day?

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MR. SCOTT: Mr. Lamek was complaining,

he was telling some of our colleagues and associates
in the press that we were getting very far behind and
he was alarmed to hear that the 12 other Counsel
might take two weeks, which was, of course, just a
third of what he had taken. So, he encourages us to
be as brief as we can to leave more time for him.
But we'll move along as quickly as I can.

THE COMMISSIONER: There's a problem

Mr. Sopinka mentioned to me that apparently he is not
available on Thursday. So, after you is Mr. Ortved
and after Mr. Ortved, I guess, is Mr. Strathy.



GG2.4

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Mr. Strathy, you are prepared to proceed?

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MR. STRATHY: Yes.

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THE COMMISSIONER: If time is available. I don't know, Mr. Hunt, are you mixed up with Mr. Sopinka's motion?

8

MR. HUNT: No.

9

MR. SOPINKA: They've got so many people over there.

10

11

THE COMMISSIONER: Well, I know, you work on quality.

12

13

And then, after Mr. Strathy, you will be prepared.

14

15

MR. HUNT: I would doubt whether that would be on Thursday in any event, just by the time.

16

17

THE COMMISSIONER: Well, no, but if it is I just would like you to be prepared. That may be all we'll get through on Thursday but it may not.

18

19

However, you haven't any thoughts on how long you'll be?

20

21

MR. STRATHY: If I'm reached on Thursday I would be very surprised if I was finished on Thursday.

22

23

THE COMMISSIONER: Yes. But don't give Mr. Hunt too much comfort or he won't be

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prepared.

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MR. STRATHY: No, I wouldn't do that.

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THE COMMISSIONER: All right.

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MR. SOPINKA: Thank you, Mr.

6

Commissioner.

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--- Whereupon the hearing adjourned until Wednesday,
August 17th, 1983 at 10:00 a.m.

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